Université de Montréal

Associations between area-level unemployment, body mass index, and risk factors for cardiovascular disease in an urban area

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Mémoire présenté à la Faculté des études supérieures et postdoctorales en vue de l'obtention du grade de Maitrise en Santé Communautaire

septembre, 2008

Ce mémoire intitulé:

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ABSTRACT

INTRODUCTION: Little is known about whether area-level unemployment is independently associated with individual-level Cardiovascular Disease (CVD) in an urban setting. Furthermore, it is unclear whether this relationship differs by sex. This thesis examined the direction and magnitude of the association between area-level unemployment (ALU) and Body Mass Index (BMI) and a marker for CVD risk, and whether this association differs by sex. **METHODS:** A sample of 342 individuals from the Montreal Neighbourhood Survey of Lifestyle and Health (MNSLH) self-reported behavioural and socioeconomic information. A registered nurse collected biochemical and anthropometric data. ALU was operationalised within a 250 m radius buffer centered on individual residence using a Geographic Information System (GIS). Generalized Estimating Equations were used to determine if body mass index (BMI), and a cumulative score for total cardiometabolic risk (TCR) representing elevated values for total cholesterol, triglycerides, high-density lipoprotein cholesterol, and glycosylated hemoglobin, were associated with ALU. **RESULTS:** After adjustment for age, gender, smoking status, behavioural, and socioeconomic covariates, living in an area in the upper ALU quartiles was associated with an elevated BMI [Q4 beta = 2.1 kg/m^2 (95% CI: 1.02-3.20)] and greater TCR [Q4 RR = 1.82 (95 % CI: 1.35-2.44); Q3 RR = 1.66 (95% CI: 1.33-2.06)] relative to the 1st quartile. Sex-by-ALU interaction revealed a 1.99 kg/m^2 (95% CI: 0.00-4.01) difference in BMI and 1.39-fold (95% CI: 1.06-1.81) greater TCR Score for women compared to men. **CONCLUSIONS:** Area-level unemployment is associated with greater CVD risk in men and women but associations are stronger among women. **KEYWORDS**: Body Mass Index; Cardiometabolic Risk; Area-Level Unemployment; Social Context

RÉSUMÉ

INTRODUCTION: Il existe peu d'évidences sur l'association entre le taux de chômage dans le milieu résidentiel (CR) et le risque de maladies cardiovasculaires parmi les résidents de milieux urbains. De plus, on ne sait pas si ce lien diffère entre les deux sexes. Cette thèse a pour objectif de déterminer la direction et la taille de l'association entre le CR et le risque de maladies cardiovasculaires, et d'examiner si cette association varie en fonction du sexe. **MÉTHODES:** Un sous-échantillon de 342 participants de l'Étude sur les habitudes de vie et la santé dans les quartiers montréalais a rapporté ses habitudes de vie et sa situation socio-économique. Des mesures biologiques et anthropométriques ont été recueillies par une infirmière. Le CR a été opérationnalisé en fonction d'une zone-tampon d'un rayon de 250 m centrée sur la résidence de chacun des participants à l'aide d'un Système d'Information Géographique (SIG). Des équations d'estimation généralisées ont été utilisées afin d'estimer l'association entre le CR et l'Indice de Masse Corporelle (IMC) et un score cumulatif de Risque Cardio-métabolique (RC) représentant la présence de valeurs élevées de cholestérol total, de triglycérides, de lipoprotéines de haute densité et d'hémoglobine glyquée. **RÉSULTATS:** Après ajustement pour l'âge, le sexe, le tabagisme, les comportements de santé et le statut socio-économique, le fait de vivre dans un endroit classé dans le 3e ou 4e quartile de CR était associé avec un IMC plus élevé (beta pour Q4 = 2.1 kg/m2, IC 95%: 1.02-3.20; beta pour Q3 = 1.5 kg/m^2, IC 95%: 0.55-2.47) et un taux plus élevé de risque cardiovasculaires Risque Relatif [RR pour Q4 = 1.82 (IC 95 %: 1.35-2.44); RR pour Q3 = 1.66 (IC 95%: 1.33-2.06)] par rapport au 1er quartile. L'interaction entre

le sexe et le CR révèle une différence absolue d'IMC de 1.99 kg/m^2 (IC 95%: 0.00-4.01) et un risque supérieur (RR=1.39; IC 95%: 1.06-1.81) chez les femmes par rapport aux hommes. **CONCLUSIONS:** Le taux de chômage dans le milieux résidentiel est associé à un plus grand risque de maladies cardiovasculaires, mais cette association est plus prononcée chez les femmes. **MOTS CLÉS**: Indice de Masse Corporelle; Risque Cardio-métabolique; situation socio-économique; taux de chômage dans le milieu residential.

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LIST OF ABBREVIATIONS

CVD: Cardiovascular Disease

CHD: Coronary Heart Disease

MNSLH: Montreal Neighbourhood Survey of Lifestyle and Health

ALU: Area-level Unemployment

ALE: Area-level Education

BMI: Body Mass Index

TCR: Total Cardiometabolic Risk

CT: Census Tract

GIS: Geographic Information System

DEDICATION

For my parents, and others who introduced me to the writings of the Bahá'í Faith.

> *Be anxiously concerned with the needs of the age ye live in, and center your deliberations on its exigencies and requirements.*
>
> *One hour's reflection is preferable to seventy years of pious worship*
>
> *Bahá'u'lláh*

ACKNOWLEDGEMENTS

Special thanks must be made to Dr Mark Daniel, for allowing me to get this project off the ground in the first place; to Dr Ian Shrier, who willingly guided me through some knotty conceptual terrain; and to Dr Catherine Paquet, whose presence, though often electronic, was most helpful.

1. INTRODUCTION

1.1 HEART DISEASE IN NORTH AMERICA

Even a cursory review of the history of public health in North America over the past century reveals that heart disease has exerted a predominating influence over the evolution and mindset of Western medicine and society. Three decades into the 20th century witnessed the first signs of the coronary heart disease epidemic—the "disease of the intelligentsia"—which was then attributed to the excessively stressful lifestyles of the professional, financial and political elite (Rothstein 2003)(p. 206). By the 1940s and 50s, coronary heart disease (CHD) was ranked the first major cause of death in North America (Levy 1981), with CHD specific mortality rates as low as 2% for Canadian women (Manuel et al 2003) and as high as 7.4% for American men (Rothstein 2003) between the ages of 65 and 74 years. Shortly thereafter, the North American medical establishment began to address the rising burden of cardiovascular disease.

Coronary heart disease is a potentially lethal development of plaque in the coronary arteries resulting in a compromise of oxygen delivery to the heart muscle. It was distinguished early on from Cardiovascular Disease (CVD)—a systemic form of CHD manifested throughout the cardiovascular system, rather than just the coronary arteries—to deal with the threat posed by the interruption of oxygen to the heart that commonly resulted in death. In order to better understand these diseases, medical practitioners began to use the concept of a 'risk factor' developed in the Life Insurance industry, as well as newly developed correlation and inferential techniques (Rothstein 2003; Stigler 1986), allowing them to establish relationships between numerous behavioural characteristics and the presence and development of cardiovascular and coronary heart disease.

One of the first studies to examine the determinants of cardiovascular disease was the Framingham Heart Study, which set out to "[focus] on arteriosclerotic and hypertensive cardiovascular disease," with the assumption that "they are the result of multiple causes … which work slowly in the individual." (Dawber et al 1951)[p. 280]. Since then, a large number of individual 'risk factors' have been uncovered that predispose individuals to cardiovascular disease (Rothstein 2003).

Yet while this research was adding valuable information to the medical arsenal, developments in other areas of epidemiology were to bring a new perspective on this burdensome disease. Epidemiologists began to highlight the fact that these new risk factors did not consider the social, political and economic contexts in which CVD unfolded. Some claimed that this search was nothing more than "occupational therapy for epidemiologists," based on misbegotten conceptions of the causes of CVD rather than a fair assessment of all contributing factors (Beaglehole & Magnus 2002). Beginning with signal publications such as John Cassell's "Social science theory as a source of hypotheses in epidemiological research" (Cassel 1964), and Geoffrey Rose's "Sick individuals, sick populations" (Rose 1985), and then propelled by a raft of articles in the mid-1990s that challenged the atheoretical nature of epidemiological research (Krieger 1994; Krieger & Zierler 1996; Link & Phelan 1995; McMichael 1995; Pearce 1996; Susser & Susser 1996; Victora et al 1997), epidemiology began to move from a strictly biomedical, individualistic approach, to one which sought to understand how biological determinants of disease unfold in the context of social, cultural, economic and political environments.

1.2 THE ENVIRONMENT AND DISEASE

That the environment plays a role in the development and progression of disease was hardly a new idea: Ancient Hippocratic texts (Hippocrates 1978), East-Asian medical compilations (Anonymous 1949), and 17th century political treatises (Petty 1969) noted the relationship between the social and physical environments and the health of populations (Working Group on Health Disparities 2005); Well known 18th and early 19th century disease theories pointed to social and physical environments as a major source of contagion (Chadwick 1965; Engels 1958; Rosen 1993; Taylor 1974; Taylor & Rieger 1985; Villermé 1829). Yet between the 1870s and the mid-1940s, a number of major scientific and world events occurred that would change the way medical science was carried out.

The advent of the Henle-Koch postulates saw the beginnings of an "epistemological revolution" (Kunitz 2007) in medical science—a shift to an individualistic laboratory based paradigm that was to become one of its defining characteristics (Carter 2003). Around the same time, massive sociopolitical pressures were channeling scientists to the production of wealth and tools of war (Bernal 1971(Vol. IV. p. 831-848); Hartcup 2000), or steering them into politically and socially "neutral" terrain (Schrecker 1986; Smith 1990; Wang 1999). As a result, and despite a minor undercurrent of literature advocating for more focus on the environmental influences on biological phenomena (Livingston 1934; Sydenstricker 1933), most medical scientists of the 1940s, 50s, and 60s embraced the doctrine of specific biological etiology, and eschewed any line of research that questioned the nature of society, and its potential impact on the population's health (Susser 1985).

It was not until the early 1960s that the first group of studies began to address aspects of the social environment in relation to CVD. These studies related occupational (Zukel et al 1959), socioeconomic (Scotch 1963), cultural (Syme et al 1964), racial (Comstock 1995), and urban form (Gampel et al 1962) characteristics to CVD risk factors and outcomes. By the mid-1980s, social aspects of CVD became important research topics. Strong evidence compelled researchers to accept that: (1) a gradated relationship existed between various socioeconomic characteristics and CVD related morbidity and mortality (Davey-Smith et al 1990; Fox 1989; Marmot et al 1987; Townsend & Davidson 1982; Wing 1988); and (2) there existed a plausible relation between socioeconomic characteristics and the development of atherosclerotic lesions (Kaplan et al 1982), altered myocardial physiology (Beamish et al 1985; Lown et al 1977), and the progression of classical CVD risk factors (Siegrist 1991). Since these developments, contemporary epidemiology has become a discipline that is quickly moving towards developing an understanding of higher-order determinants of disease—determinants such as social or economic circumstances in which pathophysiological determinants are embedded and take form. These determinants have been looked at in a number of key contexts, foremost among them being neighborhood and community settings.

However, despite the movement to focus on neighbourhood and community determinants of health and disease, there remain some key limitations in the research. For one, representations of neighbourhood and community context have been limited. Much of the research linking CVD to "neighbourhood" socioeconomic status (SES) has operationalised neighbourhood social context using an array of cumulative indices or

summary scores incorporating aggregate measures income, education, and occupation. This has led to a number of praxis-based challenges when trying to carry knowledge produced in neighbourhood studies on health into the policy shop (Krieger et al 1997; Oakes & Rossi 2003). Cumulative indices or summary scores, though valuable representations of an underlying and broad social context, may detract from tangible policy initiatives insofar as they conflate pathways and obscure the independent contributions of each component to specified health outcomes (Krieger et al 1997).

Another complexity results from the choice of components with which these indices are created. The use of education, income, and occupation each have strengths as a representation of some aspect of social context. Education is a stable indicator of socioeconomic position since education levels do not generally depend on health status of the population at a given instance (Krieger et al 1997)[p. 364]. Income is an important tool that can allow individuals access to the necessary elements of a healthy lifestyle (Krieger et al 1997)[p. 359]. Occupation, usually used as an indicator of social prestige, can be a marker for access to resources not usually available through higher wages or more education (Krieger et al 1997)[p. 346].

Yet, with each of these measures, there are important drawbacks that cannot be ignored. In some circumstances, educational stability may also be a liability insofar as the use of education precludes capturing variations in social context that may give rise to disease (Davey-Smith et al 1998a; Liberatos et al 1988).

The volatility and complexity of a variable like income is done little justice in public health studies that operationalise it with simple proxies such as median household income (Duncan 1996). Even an accurate measure of median household income

aggregated to the community-level may not necessarily be predictive of a given neighbourhood's ability to access the necessary resources for improving poor health or maintaining good health. Studies have shown that goods and services available to residents of higher-income neighbourhoods tend to be better in quality and lower in price than those available to residents of lower income neighbourhoods (Kaplan 1996; Krieger et al 1997; Macintyre et al 1993; Troutt 1993).

Occupation, when used exclusively as a prestige-based measure, does not provide information about how material aspects of socioeconomic deprivation shape patterns of population health (Wegener 1992): prestige is not a material resource one can use to improve or maintain health (Krieger et al 1997)[p. 366]. Furthermore, evidence suggests that the face validity of occupational status is inconsistent across gender (Pugh & Moser 1990)—i.e. it does not represent the same underlying concept for men and women in the same occupation.

Finally, each of these measures, though inherently linked to the social, political, and economic factors outside of the control of any one individual's agency, can still be "explained away" as individually based social variables that can be influenced by choice rather than circumstance. Like the choices that an individual makes about diet, exercise, and other health related behaviours, income, education, and occupation can be seen as no more than the culmination of life decisions—a view that relegates higher-order social determinants of health back to the realm of individual behaviour, and de-emphasizes the importance of wide-scale policy changes that could potentially influence population health. This may explain why, despite Canada's role as a leader in health promotion and recent advances in understanding the social determinants of health, Canadian health

policy has maintained its individually based 'lifestyle' approach to health promotion (Raphael 2008).

Generally speaking, these drawbacks do not apply to area-level unemployment. For one, it is difficult to construe ALU as an individualized variable, or the product of ill-advised life decisions. Area-level unemployment stands in a unique position as a marker for area-level social context since it spans the gamut of what Amartya Sen calls policy "spaces" (Sen 1997), being influenced by organizational matters beyond individualized income considerations. Area-level unemployment is tightly linked to policy decisions in the realm of urban planning (Kitchen 2001); fiscal policy and inflation (Fortin 1980); foreign trade, investment, and exchange (Gilpin 2001); and immigrant's rights, political representation of minority groups, and social polarization and inequality (Hofrichter 2003)[p. 258]. Finally, in the current global economic crisis, numerous changes are expected to occur. Among the first and most dramatic changes expected to impact Canada's economic situation, and particularly in Québec and Ontario, is record-high rates of unemployment (Anonymous Oct 9th, 2008). For these reasons, we thought it appropriate to investigate the relationship between area-level unemployment and markers for cardiovascular disease. Because the literature on cardiovascular disease and area-level unemployment is hazy with regards to specifics, we set out to address the gaps in the knowledge base.

In particular, and as we will elaborate in the pages to come, we sought to target three gaps in the literature on the association between area-level unemployment and cardiovascular disease: 1) whether or not there is an association between area-level unemployment and cardiovascular risk factors in an urban setting; whether or not this

association exists when taking into consideration the age-group most vulnerable to cardiovascular disease; and whether or not this association exists after controlling for well-defined covariates, and—for comparison's sake—after adding commonly included intermediates to our statistical models.

All in all, it is highly likely that area-level unemployment influences health by: (1) limiting access to necessary physical and social resources (Daniel et al 2008; Robert 1999); (2) through the exposure to, and appropriation of, untoward normative behaviours (Karvonen & Rimpelä 1996); and (3) by increasing allostatic loads through stressful environmental situations, leading to maladaptive regulatory shifts in insulin, lipid, oxidative and inflammatory biomarkers, and therefore altered cardiometabolic status (Daniel et al 2008). Yet despite these likely relations, only a very limited literature deals with the specific issue of area-level unemployment and health.

2. Literature Review

2.1 Area Effects on Health

Recent years have seen an upsurge of interest in research on area-level effects on health. Scientists have implicated various characteristics of the area surrounding an individual, such as the concentration or density of various businesses (Chuang et al 2005; Wang et al 2007), its "greenness" (Nielsen & Hansen In Press), accessibility to parks and areas for physical activity (Ford et al 1991; Hillsdon et al 2008; Karvonen & Rimpelä 1996; Kipke et al 2007; Parks et al 2003), and even one's distance to wealthy areas (Auchincloss et al 2007). Furthermore, characteristics of the social environment have received much attention, with a particular focus on area-level SES, and its relation to self-rated health (Brown et al 2007; Cummins et al 2005a); the presence of CVD risk factors (Davey-Smith et al 1998b; Janssen et al 2006; Shishehbor et al 2008); CVD

related behaviour (Diez-Roux et al 1999; Morland et al 2002); and CVD outcomes proper (Diez-Roux et al 2001; Franzini & Spears 2003; Riva et al 2007).

These studies have generally employed three types of empirical strategies to assess area-level effects on disease (Diez-Roux 2001). The first is to meticulously contrast the differences in health outcomes between a small number of well-defined neighborhoods, with detailed information on their social, geographical and community history (Macintyre et al 1993). At the other end of the strategic spectrum, ecological studies use rates of disease and exposure occurrence in a large-scale environment (county, province, nation, state, etc.) to document large-scale trends in covariation between them. Finally, multilevel studies use information obtained at state, county, provincial, or municipal levels linked to individuals and determine whether relationships exist between the environments assessed and the outcomes documented while accounting for individual-level characteristics. With respect to CVD, ecological and multilevel studies have most often been the methods of choice in area-level empirical settings (Riva et al 2007).

2.2 Unemployment & Population Health Rates—Ecological Studies

Brenner was among the first to analyze the ecological CVD-unemployment relationship using time series analyses of national unemployment levels and CVD specific mortality rates in the U.S. (Brenner & Mooney 1983), England and Wales (Brenner 1979), Scotland (Brenner 1987a), and Sweden (Brenner 1987b). In these studies, he found positive associations between unemployment and CVD specific mortality, even after accounting for covariates such as tobacco, alcohol and dietary fat consumption trends. Adams (1981) conducted a similar analysis on Canadian data and

found a positive association between aggregate unemployment and CVD mortality, but an inverse association for overall mortality (i.e., greater unemployment associated with lower overall mortality). Similarly, Bunn (1979) found a positive association between unemployment and CVD specific mortality in an aggregate Australian study. Between 1988 and the mid-2000s, ecological studies assessing the relations between unemployment (Crombie et al 1989; Starrin et al 1988) or duration of unemployment (Starrin et al 1990) and area-wide cardiovascular drug sales (Gorecka et al 2005; Öreberg et al 1992) or age-standardized CVD mortality rates (Filate et al 2003; Öreberg et al 1992) all produced similar findings—higher unemployment rates associated with a greater presence of CVD risk markers.

Yet, as noted by many authors, the implications of these studies are not clear. Not only are inferences drawn from these types of studies prone to the ecological fallacy (Diez-Roux 1998), but a number of researchers have questioned the value of these findings on methodological (Cook 1985; Gravelle et al 1981; Kasl 1979; 1982) and substantive grounds (Cohen & Felson 1979; Colledge 1982; Gerdtham & Ruhm 2006; Ruhm 2000). For instance, in his methodological critique, Gravelle et al (1981) highlight a number of inconsistencies and contradictions in the way Brenner operationalised income, economic growth, and welfare expenditure—torts that, once remedied, lead quite easily to equally plausible but contrary conclusions. Furthermore, in the same analysis, Gravelle et al criticize Brenner's omission of key variables representing insurance coverage and improvements in medical treatment during the 40-year period of his study—variables that likely contributed to national health rates, and

therefore, that would have changed the nature of Brenner's results (Gravelle et al 1981)[(p. 676)].

Furthermore, Brenner's findings have recently faced a substantive challenge in Ruhm's (2000) analyses. Using identical methods, Ruhm showed that rather than being countercyclical—meaning that increases in unemployment coincided with decreases in national mortality—the relationship was procyclical. His conclusion was that recessions (which lead to a rise in national unemployment) led to lower overall mortality and lower cause-specific mortality in eight of the ten cause-specific mortality indicators assessed. Despite these contradictory findings, one thing is certain: given the inferential limitations associated with ecological analyses, sound policy decisions cannot be made on the basis of these studies alone.

2.3 Area Unemployment and Individual Health—Multilevel Studies

Just as the relationship between area-level unemployment and rates of CVD is unclear, there are conflicting findings about whether area-level unemployment is associated with individual health outcomes. This is true for outcome measures that are self-rated, or more objective empirically assessed measures.

Bosma et al (2001) sought to understand the relationship between neighbourhood SES and all-cause mortality in a longitudinal analysis of 8,506 men and women (aged 15-74) chosen from 86 neighbourhoods in the city of Eindoven in the Netherlands. A continuous and quartiled neighbourhood SES index was created, using percentage of subjects who reported being unemployed or disabled. The authors controlled for age, sex, baseline health, and an individual SES marker that covered social, psychological, behavioural and housing condition dimensions. The authors found that a 10% increase

in the neighbourhood proportion of subjects reporting that they were "unemployed or disabled" corresponded to an odds ratio of 1.36 (95% CI: 1.05, 1.66) for mortality after correcting for individual level SES.

In another longitudinal study, Veugelers et al (2001) evaluated the relationship between independent neighbourhood characteristics (income, education level, unemployment) and mortality in the province of Nova Scotia. Geo-linked Census information was used to derive neighbourhood level information. The unemployment rate was divided into tertiles (less than 10%; 10-15%; greater than 15%) and adjusted for age and gender (model 1); and age, gender, smoking status, Body Mass Index (BMI) and diabetes (model 2). They found no substantial or significant differences in mortality between neighborhoods with lower and higher rates of unemployment, and concluded that Canadian tax structures and the health care system may explain their lack of evidence when contrasted to the American studies they reviewed.

In 2002, Béland et al examined whether or not the relationship between perceived health and individual unemployment depends on the context of area-level unemployment in Quebec (Béland et al 2002). Analyzing data obtained from a 1987 health survey of 9,422 individuals 15 years and older, using unemployment data from the 1986 Canadian Census linked to individuals, and controlling for individual level (age, gender, perceived stress, socioeconomic resources, social network, perceived social support) and contextual (gender distribution, age-group distribution, education, proportion of immigrants, family structure, income, employment status, and occupational status) influences, they found no relationship between ALU and perceived

health, measured as a five-category health question asking people to rate their health compared to others their age.

Curtis et al (2004) used a lifecourse approach to ascertain whether area-level historic characteristics were associated with mortality in a retrospective analysis of data from the Office for National Statistics Longitudinal Study for England and Wales. Information on 62,719 individuals who were 0-16 years of age in 1939, were still alive in 1981, and whose 1939 place of residence could be verified was analyzed to evaluate the lifecourse effects of physical and socioeconomic place. Individual, compositional, and contextual variables were created for two time periods: 1981 and 1939. These variables included a number of physical, social and individual characteristics, including unemployment rates in 1939 and 1981. They found that individuals who lived in areas with higher rates of unemployment in 1939 had significantly higher rates of death between 1981 and 1991 (RR = 14 to 15%), even after controlling for individual characteristics, region of residence in 1981, and area-level SES conditions in 1981. The authors did not find significant associations for other area-level 1939 variables.

Using data from standardized health survey questionnaires, postal questionnaires investigating neighbourhood social networks, and data from service providers and government statistics in England and Scotland, Stafford et al (2005) assessed the gender differences in relationships between neighbourhood environment and self-reported health. With a sample size of 8,440, they found statistically significant interactions between gender and integration into wider society, left-wing political climate, physical quality of the neighbourhood environment, and the unemployment rate after adjusting for age, family type, individual social class and economic activity. Neighbourhood

unemployment was defined at the level of postal code sectors and divided into tertiles. Women in the highest tertile were 1.48 (95% CI: 1.21-1.81) times as likely to report poorer health, whereas men in the highest tertile were 0.95 (95% CI: 0.78-.16) times as likely to report poorer health, than those in the lowest tertile.

In a separate report of the same study (13,899 men and women, same methods), self-reported health was found to be associated with the unemployment rate, with an OR of 1.43 (95% CI: 1.23-1.66; high vs low unemployment rate), and an OR of 1.27 (95% CI: 1.10-1.47; high vs low unemployment rate) after being adjusted for sex, age, social class and economic activity (Cummins et al 2005a).

Using the 1998 Health and Social Survey of Quebec, Zunzunegui et al (2006) examined the relationship between community level unemployment and the physical and mental health of the immigrant population in 49 police districts in Montreal. Outcomes included self-rated health, BMI, and psychological distress. Independent variables included: community unemployment operationalised at the police district level and computed as the ratio of unemployed individuals 15+ years of age to individuals 15+ years of age in the labour force; age; gender; immigrant status, operationalised as "first generation immigrants" (born outside of Canada and neither French nor English as mother tongue), "second generation immigrants" (born in Canada, but whose mother tongue was neither French nor English), and "non-immigrants" (born in Canada, whose mother tongue was French or English); occupational status (unemployed, students, housewives, retired, and those on social assistance compared to those actively engaged in the workforce); household type; education; household income; a social support index measured as a score of social participation and integration, satisfaction regarding social

interactions, and social network size; a food security index providing information on food supply restrictions; physical activity; and smoking status. Using variance decomposition analysis, and after controlling for individual unemployment, immigration status and individual risk factors, they found no significant associations between community unemployment and health outcomes. The magnitude of association for each outcome variable was small and significant only for self-rated health ($p = 0.046$) and psychological distress ($p = 0.038$).

In a longitudinal study assessing the effects of individual unemployment and cardiovascular risk status in a sample of approximately 700 Swedish men, Henriksson et al (2003) found a positive and statistically significant association between individual unemployment and BMI, and individual unemployment and total serum cholesterol at baseline. BMI remained significantly associated with individual unemployment at baseline after adjustment for education and ethnicity, but including behavioural risk factors in the model rendered the association non-significant. Cholesterol's association remained significant at baseline, even after adjustment for individual SES and behaviours. At years three and six of the follow-up period however, the association between individual unemployment and BMI, and individual unemployment and total cholesterol disappeared. The authors explained these changes by pointing to increases in national Swedish unemployment rates, concluding that associations between cardiovascular risk and individual unemployment "[vary] with the unemployment rate" (Henriksson et al 2003)$^{(p.\ 305)}$. However, they did not mention whether a formal statistical test of the CVD-unemployment relation which took area-level rates of unemployment into consideration was carried out, pointing to the possibility that the

implied "effect" of the national unemployment rate may have merely been a coincidental observation.

Sundquist et al (2006) analyzed the association of neighbourhood unemployment and the incidence of CHD before and after adjustment for age, income, employment status and marital status in Stockholm County residents aged 35-64 years. The final sample consisted of 336,295 men and 334,057 women who were followed for a period of one-year. Neighbourhood unemployment was based on the proportion of unemployed individuals in small, administratively defined geographic areas, geocoded to participant's homes, and operationalised in quartiles. They found unadjusted odds ratios of 2.05 (95% CI: 1.62-2.59) and 1.50 (95% CI: 1.28-1.75) for the highest quartile of neighbourhood unemployment (compared to the lowest) for women and men respectively. Odds ratios for women decreased to 1.87 (95% CI: 1.46-2.39) but remained at 1.51 (95% CI: 1.29-1.77) for men after adjustment for age, income, employment status and marital status.

In a study that used physiologically rigorous criteria for cardiovascular disease status, Petersen et al (2006) assessed the association of preclinical vascular disease and community SES in a sample of 230 untreated hypertensive men aged 40-70 years. Preclinical vascular disease was defined using carotid artery ultrasonography and comprehensive criteria incorporating measures of intima media carotid artery thickness, and a 12-point graded atherosclerotic plaque index containing information on the number and size of deposited plaques. Community SES was defined at the Census Tract (CT) level as a combined index of median household income; percentage of households on public assistance; percentage of unemployed adults; percentage of households

beneath the federally designated poverty limit; median values of owner-occupied housing units; median gross rent; and proportion of residents 25 years and over with less than a high-school education. They found that community disadvantage was associated with odds of 1.51 (95% CI: 1.13-2.02) for plaque occurrence, even after adjustment for age, systolic and diastolic blood pressure, fasting blood glucose levels, and individual income and education (adjusted OR: 1.68, 95% CI: 1.18-2.38). No mention was made as to whether the percentage of unemployed adults was associated with plaque occurrence independent of other area-level characteristics.

Finally, in an international cross-sectional comparison of independent study samples from the German and Czech Republic, Dragano et al (2007) assessed the differential association between CT level unemployment rates and cardiovascular disease risk factors using data from nine different towns in the two countries. Samples of 4,032 German residents and 7,552 Czech residents were included in the final analysis. Outcomes included obesity, hypertension, current smoking, and low physical activity. Socioeconomic status was operationalised according to individual education. Neighbourhoods were defined by pre-existing administrative boundaries. Neighbourhood unemployment rates were divided into quartiles and adjusted for age and sex (model 1), age, sex and education (model 2), and age, sex, education, economic activity, and social isolation (model 3). They found that: (1) in model 1, significant associations existed between neighbourhood unemployment and obesity, smoking and low physical activity in both countries, with slightly stronger associations in the German cohort; (2) adjustment for education in model 2 weakened some of the associations, and rendered the association with BMI non-significant in the Czech cohort. Furthermore,

gender differences were observed for the obesity-neighbourhood unemployment association, with German women having stronger associations than German men.

3. PROBLEM STATEMENT

3.1 KEY ISSUES IN AREA-LEVEL EFFECTS ON HEALTH

This review of the literature highlights the status of current knowledge on the relationship between area-level effects—particularly area-level unemployment—and health. The results outlined in the previous review provide some evidence that area-level social deprivation is associated with negative health related phenomena: the measures of association were at or above the null. However, these measures, though consistent, were modest at best, and did not go beyond a relative risk of two, once adjustment for covariates was made. As pointed out by Daniel et al (2008), this could be due to the fact that individual-level parameters account for much of the place-health association. Yet it may also be the result of an unclear relationship between the operational units (administrative boundaries) and theoretical constructs ("neighborhood") used to determine the status of place-health relationships in epidemiological models. Thus, in attempting to assess the relationship between place and health, this review reveals at least three key issues that merit some attention. The first rests on the operationalisation of unemployment rates based on pre-defined administrative boundaries; the second, on the selection and integration of covariates in statistical models in order to assess the effects of ALU on health; and the third issue involves gender differences in area-based health effects.

3.1.1 NEIGHBOURHOOD SCALE & HEALTH

In neighbourhood studies on health, the most common technique used in the analysis of the area-level effects on CVD is to incorporate aggregate census or postal code based information into multilevel models to assess the importance of place-based factors in the incidence and prevalence of CVD and CVD related states (Cooper 2004; Diez-Roux 2001; Diez-Roux 2007). Yet, there is a growing awareness of the insufficiencies and the inextricable interpretive complexities faced when reifying aggregate measures as neighbourhood constructs that meaningfully represent particular geo-spatial areas. In addition to the difficulties of causal inference based on interpretations of statistical associations between clustered group phenomena and health outcomes (Kaufman 2005; Larsen & Merlo 2005; Oakes 2004), there is the fundamental question of construct validity for a given area-level unit. For instance, Daniel et al (2008)[p. 118-119] refer to the problematic use of pre-defined area units given the "unclear correspondence (isomorphism) between administrative measures of place and the theoretical construct ('neighbourhood') ostensibly represented". A critique by Messer (2007) and subsequent rebuttal by Diez-Roux et al (2007)[p. 872] highlights the fact that "A major challenge is developing theoretical models of the processes through which neighbourhoods (or areas) may affect health." Diez-Roux (2007)[p. 7] further expounds upon the issues of spatial scale, stating that one of the more fundamental problems is that "there is still relatively little theory on the spatial scale likely to be relevant to a specific health outcome."

The use of rather arbitrary area-level units such as CTs, postal areas or police districts stems primarily from pragmatic concerns—in particular, the availability of these respective administrative datasets. Yet using fixed boundary areas may detract

from the analysis insofar as it does not account for exposures of individuals living on the margins of the given boundary (Chaix et al 2005). These analyses are also prone to the modifiable areal unit problem (Openshaw 1984), where outcomes tend to depend on the size and shape of the selected neighbourhood (Chaix et al 2005; Fotheringham & Wong 1991; O'Campo 2003). And, as previously mentioned, outcome scale-dependence on neighbourhoods defined by administrative boundaries gives rise to many interpretive and methodological difficulties when assessing the effects of contextual deprivation (Chaix et al 2005). Taken together, these difficulties create issues of misclassification bias in utilizing administratively fixed areas as proxies for neighbourhoods, and render questionable the utility of numerous investigations on contextual deprivation and health. One way to improve this problem is the implementation of *moving-window areas,* or the designation of circular areas centered on individuals in given buffer zone with a specified radius (Chaix et al 2005), however, no studies on the relationship between ALU and CVD have yet utilized this approach.

3.1.2 Intermediate Variables & Covariate Selection

Another issue that arises involves the question of covariate selection in analyses of area-level influences and health outcome. This issue has been especially problematic in studies on the effects of place on health. For example, as Macintyre et al (2002)[p. 129] point out, "the individual controls introduced into multivariate analysis may well be intervening variables on the pathways between place and health, not 'confounders' as they are so often treated."

Yet despite their importance in neighbourhood studies, debates on how to adequately select covariates are not limited to neighbourhood studies alone. Traditional

strategies used in epidemiology to select a set of covariates have long been criticized on numerous fronts, including their poor sensitivity (Greenland 1989), their tendency to introduce bias (Weinberg 1993), and their tendency to neglect the implications of relational attributes between selected covariates (Greenland et al 1999). Such procedures generally consist of using automatic selection procedures (e.g. stepwise regression); using an arbitrary percentage change as a selection criterion when a given covariate is included in a model; or combining theory and/or background substantive knowledge with statistical associations to select particular covariates (Hernán et al 2002). These strategies, despite their pervasive use, often lead to bias due to the exclusion of overlooked yet pertinent confounding variables or the inclusion of unnecessary confounders (Greenland 1989; Greenland et al 1999; Greenland & Robins 1986; Pearl 2000; Robins 2001; Weinberg 1993).

Furthermore, the issue of covariate selection is not inconsequential. Formal recognition of the inclusion of covariates that lie on the causal pathway between area-level characteristics—such as ALU—and outcomes of interest—such as mortality or markers of morbidity—may result in significant changes in the nature of the conclusions drawn from studies on neighbourhood effects on health. For example, Veugelers et al, after adjusting for age, gender, BMI and diabetes in their assessment of the effects of neighbourhood SES on mortality, stated that the difference in the relative risk of their study (RR = 2.2) compared to that of Yen & Kaplan (RR = 5.5) may have been due to the "free access to basic health care and more access to other public goods" (Veugelers et al 2001)$^{(p.\ 730)}$ that characterized their Nova Scotian cohort. However, they neglected to address the fact that in the relative risk that they used as a comparison, Yen & Kaplan

only adjusted for smoking and perceived health status and did not include BMI or diabetes as covariates in their model (Yen & Kaplan 1999). Any effect of neighbourhood SES on mortality that would have acted through BMI and diabetes would have been accounted for in the analysis by Veugelers et al, thus removing bias from the point estimate where these correlates of mortality occurred more frequently in disadvantaged areas. This was not the case with Yen & Kaplan's relative risk of 5.5, and recognition of this fact may have led Veugelers et al to use a relative risk more germane to their own analysis as a basis for comparison.

One potential solution to the problems faced in covariate selection is to use graphical tools to frame the relations between pertinent variables in order to justify selection of covariates, and even link potentially causal relations to statistical associations (Rothman et al 2008). A number of logico-mathematical covariate selection criteria based on graphical analyses have been developed which correspond to traditional criteria for identifying confounders in epidemiological data (Glymour 2006). Furthermore, some have argued that this technique surpasses conventional rules for confounding identification because of its ability to avoid the introduction of bias due to over-adjustment (Rothman et al 2008)[p. 194-6]. In this study, an *a priori* graphical model was used to represent relational attributes of numerous variables within the context of a specified conceptual framework. This model was then reduced to two empirical analytical models that served to guide statistical analyses.

3.1.3 Gender Differences in Area-Level Effects

The literature review also revealed that some studies found higher risk for women than for men living in the same stratum of area-level unemployment (Dragano et

al 2007; Stafford et al 2005; Sundquist et al 2006). These findings coincide with others that have shown similar gender differences in the degrees to which environments are associated with the cardiovascular status of men and women (McKinlay 1996; Molinari et al 1998). Given these known differences, it is important to consider gender differences in area-level unemployment on cardiovascular disease. Therefore, we will explore whether our associations will differ according to gender, accounting for relevant confounders and behavioural covariates.

3.2 Research Question and Conceptual Framework

We set out to assess whether ALU, as a marker for area-level socioeconomic and physical characteristics, was related to the antecedents of CVD—namely, BMI and a marker for total cardiometabolic risk. Specifically, our aims were to answer the following research questions:

1. Is ALU associated with an elevated BMI and a higher total cardiovascular risk when considering the perceptually relevant spatial area centered on the individual?
2. Does this relation hold after accounting for necessary covariates determined using a Directed Acyclic Graph, and including individual behaviour, individual SES, and an alternative marker of area-level SES?
3. Do any of these associations differ by gender?

The conceptual framework (Figure 1) within which we will be working is derived from Daniel et al (2008)[p. 117] and outlines the "pathways by which the geospatial clustering of disadvantage might be viewed as causally related to cardiovascular and glycemic disease." In this framework, ALU stands as a *risk*

condition—an indicator of the structural and contextual characteristics of the environment that represent an asymmetrical exposure to economic, social, and political infrastructure, as well as to collective norms and behaviours, resulting in a differential distribution of health and disease in the population. These risk conditions influence the biological antecedents of disease (1) directly, through non-conscious perceptions and increases in allostatic load, and (2) indirectly, through psychosocial factors which pattern lifestyle and health behaviour.

This conceptual framework was used to create a Directed Acyclic Graph (DAG) positioning various constructs in a causal network depicting relations pertinent to our research questions (Figure 2). Two reduced analytical graphs illustrating the potential causal relationships between variables on which we had information were then derived from this general graphical model: one for BMI and one for total cardiometabolic risk (TCR). Given that the etiological relationships between our variables under consideration were similar for both BMI and TCR, we combined both models into one. Thus Figure 3 represents the graphical causal model for the relationships between the chosen set of covariates and both outcome variables.

Even though standard covariates based on our DAGs were limited to age and area-level education, we decided to include behavioural and individual socioeconomic covariates as well. Although they do not meet the standard confounder definition, we decided to include them in separate models (Models 3 and 4) in order to better compare the results from our studies to other studies on neighborhood effects on health, which tend to include such intermediates. Thus results are presented in 4 models, the first two

including standard confounders, and the last two including standard confounders as well as variables commonly included in studies on area-level effects on health.

With these research questions and this conceptual framework, we hope to address specific gaps in the literature on area-level unemployment and cardiovascular disease. Of the total of three studies that have looked at the association between ALU and CVD, two have not accounted for behavioural variables in their measures of association (Dragano et al, 2007; Sundquist et al 2006), one omitted the age group of individuals most vulnerable to CVD events associated with an elevated BMI (Dragano et al, 2007), and one operationalised unemployment at the level of police districts and found null effects (Zunzunegui et al, 2006). We set out to address our research questions (is there an association between area-level unemployment and CVD risk factors in an urban setting? Does the nature of this association change after adjusting for intermediate variables often included in statistical models? Does this association differ according to sex?) while taking into account each of these issues.

4. METHODS

Data for this study were collected from the Montreal Neighbourhood Survey of Lifestyle and Health (MNSLH). Our objectives were to collect detailed information on self-reported health and biological measures integrated into a Geographic Information System (GIS) [Daniel M, Kestens Y. MEGAPHONE(®1046898): Montreal Epidemiological and Geographic Analysis of Population Health Outcomes and Neighbourhood Effects].

4.1 SAMPLING

We set out to investigate the relationship between area-level contextual measures, individual behavioural measures, and biological outcome measures using a stratified cluster sampling design. The sampling frame for the field study component included 521 census tracts (CTs) on the Island of Montréal stratified into low, medium and high SES categories based on an index combining educational attainment and income. Using the 2001 Census, we then randomly selected CTs with higher proportions of French and English speakers, and subsequently matched them on socioeconomic variables. Six census tracts were selected from this stratified cluster design; one additional medium-SES French tract was later added to augment sample size.

Recruiters canvassed all non-commercial addresses within the selected CTs, leaving informational materials in residential mail-boxes. This delivery was followed up within 48 to 72 hours by a home-visit in order to inquire about the willingness to participate. Up to four follow-up attempts were made and a note was left for individuals absent at the time of visit inviting them to contact the researchers. During the follow-up, participants were invited to fill out the questionnaire via a scheduled phone interview, online, or on paper. Between 4,200 and 4,900 envelopes with informational materials were distributed within the 7 designated CTs. A sample of 131 addresses was used to examine the response rates. Of the 131 addresses, 55 individuals were interviewed and two were non-eligible. Of the 53 eligible individuals, 19 agreed to participate, resulting in a 14.5% estimated response rate, and a 36% estimated cooperation rate.

The final sample consisted of 415 individuals with a mean age of 34.73 years, 209 of which were female and 206 of which were male. The number of individuals

sampled from each of the seven CTs was 22, 37, 46, 62, 70, 85, and 93. Of the 415 individuals recruited for our study, 73 were excluded due to missing information. Four had missing age information, five were missing information on alcohol consumption, seven on physical activity, seventy one were missing BMI information and 73 TCR information leaving us with a final sample of 342 individuals.

4.2 Collection of Area-level Information

Area-level and contextual information was derived from the 2001 Canadian Census incorporated into MEGAPHONE©, which integrates a large variety of additional geo-coded databases pertaining to area-based socio-demographic and socio-environmental indicators. Participant's residence were geocoded at the address level and geolinked to census data. Using these data, we created buffers with a 250 m radius centred on individuals' homes using GeoPinpoint© Software (DMTI Spatial). Two hundred and fifty meters was chosen to represent immediate neighbourhood influences. Weighted averages based on the proportions of different census tracts contained in the 250 m buffer zone for a given variable were calculated and used as our independent area-level variables (Figure 4). These aggregate variables coded with the GIS served as the independent variables and covariates in our statistical analysis.

4.2.1 Area-level Covariates & Main Effect

The unemployment rate of the environment immediately surrounding each individual was calculated using information obtained from the 2001 Census integrated into MEGAPHONE. The Census-based definition of unemployment "refers to persons 15 years and over, excluding institutional residents, who, during the week (Sunday to Saturday) prior to Census Day … , were without paid work or without self-employment

work and were available for work and either: (a) had actively looked for paid work in the past four weeks; (b) were on temporary lay-off and expected to return to their job; (c) had definite arrangements to start a new job in four weeks or less." (Statistics Canada 2007). A 250 m buffer-zone centered on the individual's place of residence was created, and the census-based unemployment rate within this zone was then geo-linked to the individual. If the buffer-zone fell entirely within a given Census Tract, then the Census unemployment rate corresponded exactly to the buffer-zone rate. If, however, the buffer-zone overlapped 2 or more Census Tracts, then a weighted average of the unemployment rate in each Census Tract which the buffer-zone overlapped was calculated based on the overlap size (Figure 4). The same technique was used to calculate area-level education. Area-level education was operationalised using the proportion of the population 20 years and older with less than grade 9 as their highest level of education (Veugelers et al 2001). Finally, given that neighbourhood effects are likely nonlinear (Granovetter 1978; Sundquist et al 2006), we modeled area-level variables categorically. In order to increase the discriminative ability of our main effect, ALU was operationalised using quartiles. Area-level education was operationalised into categories based on gaps in the variable's distribution.

4.3 Collection and Validation of Individual Information

In order to reduce the overall respondent burden often associated with population-based studies, the MNSLH protocol allowed participants to complete questionnaires by telephone, Internet, or hard copy mail-outs, with in-home collection of anthropometric data by registered nurses. We gauged the generalizability of our sample

by systematically comparing it to the 2001 Canadian Census population using Fisher's double-sided exact probability test.

4.3.1 BEHAVIOURAL COVARIATES

Behavioural measures included diet, physical activity, smoking status, and alcohol intake. We used a variation of the International Physical Activity Questionnaire inquiring about the number of days per week that participants 1) walked at least 10 minutes at a time, 2) walked *specifically* to maintain their health or fitness, and 3) performed at least one vigorous activity for at least 10 minutes during their leisure time. Using a standard formula (IPAQ, 2005), the questionnaire information was converted to the number of Metabolic Equivalents (METS) expended over the previous seven days. The METS score was then converted to a standardized score. These instruments (questionnaire and conversion formula) have produced Spearman's ρ reliability and validity estimates of 0.8 and 0.3 respectively, comparable with most other self-report validation studies (Craig et al 2003).

A modified version of the U.S. Behavioural Risk Factor Surveillance System questionnaire was used to assess fruit and vegetable consumption (Pérez 2003; Serdula et al 1993). The questionnaire response options were 1) none, 2) 1 day, 3) 2-3 days, 4) most days, and 5) every day, and respondents were asked to choose which option best represented their consumption of eight different sets of fruits and vegetables over the past seven days. In addition to the 6 BFRSS questions, the MNLSH added questions about frozen fruit and frozen vegetable consumption. A total fruit and vegetable consumption score was calculated based on the sum of responses to the eight questions and operationalised as a continuous variable. Furthermore, fast food consumption was

estimated using the number of times a fast food restaurant (FFR) was visited in the previous week as a proxy for consumption. This score was dichotomized using a cutoff of one or more FFR visits in the previous week based on a gap in the distribution at this level. A score of zero was used as referent.

Smokers were originally categorized as non-smoker, former smoker, light smoker and heavy smoker, based on the number of cigarettes smoked in the previous week (Beck et al 2005). However, initial analyses revealed no differences between the non-smoker/former smoker groups, and between the light-smoker/heavy-smoker groups. Therefore, these two categories were combined to create one dichotomous smoker/non-smoker variable with non-smoker as referent.

Alcohol consumption was measured via a questionnaire that asked respondents about the quantity of alcohol they consumed over the previous week. Responses were then categorized as "abstainer," "light drinker" (no more than 1 drink per day for women and two drinks per day for men) and "heavy drinker" (more than 1 drink per day for women and two drinks per day for men) based on the 2005 United States Department of Agriculture and Department of Health and Human Services Dietary Guidelines (United States Department of Agriculture 2005). Abstainer was used as the referent.

4.3.2 Individual-level Socioeconomic Covariates

Individual-level socioeconomic covariates included education, income and employment status. Age was categorized as a continuous variable. Male was referent for the gender variable.

Individual income was assessed using a nine-point question asking respondents about the total yearly income for their entire household. This question was operationalised using 2 dummy variables for total yearly household income between (1) $20,000 and $50,000, (0 = referent) and (2) $50,000 plus (0 = referent).

Education was assessed using a similar nine-point question asking respondents about the highest level of education obtained, and operationalised as a dichotomous variable. In order to approximate a correspondence across individual- and area-level education measures, individuals with greater than or equal to a high-school education were used as the referent.

Employment status was determined by asking respondents whether they were currently: (1) students; (2) homemakers; (3) unemployed and looking for work; (4) on sick leave; (5) on maternity leave; (6) self-employed; (7) working part-time; (8) working full-time; or (9) retired. This variable was then used to operationalise individual unemployment by creating a dichotomous variable. Unemployed status was used as the referent.

4.3.4 Dependent Variables

Biological markers were measured via finger-prick blood samples, collected from each individual by a registered nurse and analyzed for blood cholesterol, blood lipid profile, and glycosylated haemoglobin (HbA_{1c}; LDX cholesterol, and GDX hemoglobin A1c analyzers, Cholestech, Hayward, CA). Coefficients of variation for blood cholesterol, blood lipid, and glycosylated haemoglobin measurements ranged between 2%-5% (Cholestech, 2002a; Cholestech, 2002b). These measures provided data for one of the dependent variables. A clinically relevant index for total cardiometabolic

risk was created by summing the number of cardiometabolic indicators that exceeded a clinically relevant cut-off value. These indicators included glycosylated hemoglobin (HbA_1c; measured as a percentage), triglycerides (TRG; mmol/L), total cholesterol (TC; mmol/L), and high density liproproteins (HDL; mmol/L). The cutoffs used were: $HbA_1c \leq 7.0$ %; TRG ≤ 1.7 mmol/L; TC ≤ 5.0 mmol/L; HDL ≥ 1.29 mmol/L for women and 1.03 mmol/L for men based on the American Heart Association Guidelines for Primary Prevention of Cardiovascular Disease and Stroke (Pearson et al 2002). Each variable was dichotomized based on these cutoffs, and dichotomous scores were summated to calculate a total cardiometabolic risk score with a range of zero to four.

Anthropomometric measures, including weight and height were taken by a registered nurse and used to estimate Body Mass Index (BMI) according to the standard formula (weight in kg / height in m^2). BMI was retained in its continuous form. BMI and TCR were considered separately to ascertain whether area-level effects differentially influence anthropometric and hematological CVD antecedents.

All participants gave their informed consent prior to their involvement, and the study protocol was approved by the Human Research Ethics Committee of the Centre de recherche du Centre Hopitalier de l'Universite de Montreal (CR-CHUM) and the Human Research Ethics Committee for the Social Sciences at McGill University.

4.4 STATISTICAL ANALYSIS

Analyses were carried out using SPSS 14 (SPSS 2005). Generalized Estimating Equations (GEEs) were used to model the effects of area and individual level predictors on BMI and Total Cardiometabolic Risk (TCR) to account for clustering within CTs.

We chose to model associations using GEE rather than Multilevel Modeling due to our limited number of clustering units (CTs). The GEE technique is commonly used to analyze correlated data, where treatment of individual observations as independent observations would result in underestimated standard error and confidence interval estimates, imprecise parameter estimates, and the inflation of type I error (Blalock 1984; Diez-Roux 1998; Hanley et al 2003; Kobetz et al 2003). Associations for BMI were assessed using the identity link function and normal error distribution, with results expressed as beta coefficients. A Poisson regression model fit was used to assess associations for TCR (a count measure) and are expressed as relative risks (RR). CTs were used as the clustering unit, with an exchangeable type correlation matrix.

Four statistical models were used to ascertain the relation between ALU and BMI, and ALU and TCR after correcting for necessary covariates, defined by our graphical models. Covariates were introduced in blocks. Model 1 was the baseline model and included variables that fit criteria for a confounding variable, (Rothman et al 2008)[p. 132] and included age and gender. Model 2 included the Model 1 covariate block as well as an alternative indicator for area-level socioeconomic status, area-level education. These two models contained what we were able to define as confounders.

Model 3 included the confounders in two previous blocks as well as individual level socioeconomic variables, including individual education, employment status, and income. Finally, Model 4 adjusted for individual-level behavioural variables in addition to previous blocks, including fruit and vegetable consumption, fast food consumption, physical activity, alcohol consumption, and smoking.

Once all four models were run for TCR, a sub-component analysis was carried out in order to determine the contribution of each TCR component to the overall TCR effect. Model's 1 through 4 were run on each dichotomous TCR sub-component (HDL, TC, TRG, HbA_1c) using GEEs with a Binomial probability distribution and a Logit link function. As such, results are expressed as Odds Ratios.

Model diagnostics included Pearson Residuals plotted against the Predicted Value of the Linear Predictor (Norušis 2006). Separate scatterplot panels were used to visualize variables from different CTs. Outlying variables were identified visually based on their distance from the plot cluster. Analyses were re-run with outliers removed. No outliers were visible in the TCR model. Four outliers were identified and removed from the BMI model. However, the results did not differ between the BMI models with and without the outliers, and therefore all analyses were performed with complete data. Assessment of the Variance Inflation Factors (VIFs) indicated that no multicollinearity in the predictor variables was present.

5. RESULTS

5.1 DESCRIPTIVE STATISTICS

Demographic, behavioural, socioeconomic and biological characteristics are presented by gender, and summarized in Table 1a. Table 1a indicates that, in general, women had similar BMIs but a more favourable TCR profile relative to men. Relative to women, men exercised more, frequented fast food establishments more often, had greater unemployment, smoked more, and consumed more alcohol. Table 1b outlines the mean and range of unemployment within each ALU quartile. Table 1c presents age- and sex-adjusted descriptive statistics for ALU by both outcomes and each component of the TCR score. Finally, Table 2 indicates no observable difference between 20

demographic and socioeconomic variable proportions in our sample and the 2001 Census population except for age, education, and marital and immigrant status.

5.2 Main Association

Table 3 indicates the associations between ALU and both BMI and TCR from the overall analysis (male and female). A gradated relationship was apparent between ALU quartiles and both BMI and TCR. This relationship was unchanged after accounting for area- and individual-level covariates.

5.2.1 Body Mass Index

For men and women together, there was a monotonic relationship between BMI and ALU, greater ALU being associated with greater BMI. Relative to the first quartile, this relationship was statistically significant for quartiles 2-4 in models including age and area-level education. For quartiles 3-4 these associations were unchanged upon the inclusion of individual education, income and employment status (Model 3) and behavioural covariates (Model 4).

Analysis of Pearson Residuals vs Predicted Value of the Linear Predictor scatter-plots revealed four outliers (ID: 102, 177, 199, 226) that were subsequently removed. Data were re-analyzed and negligible changes were observed in the relationships between ALU and BMI. However, these changes did not significantly alter the general patterns observed between ALU and BMI, or the conclusions drawn there from.

5.2.2 Total Cardiometabolic Risk

Similar to the BMI analysis, the association between TCR and ALU was significant and gradated after controlling for the sequence of covariate blocks. For quartiles 2-4, associations were unchanged upon the inclusion of age, area-level

education, and markers of individual socioeconomic status when compared to the first referent quartile. Quartiles 3-4 remained significant after the addition of behavioural covariates into the model. Model diagnostics revealed no outliers in the Pearson Residuals vs Predicted Value of the Linear Predictor plots.

Sub-component analysis revealed an increase in the magnitude of association after adjustment for the three series of covariates in all components except Total Cholesterol. Furthermore, as Table 4 demonstrates, in Model 4, the association was strongest for HbA_1c (OR = 7.45, 95% CI: 3.78-14.68), followed by TRG (OR = 4.51, 95% CI: 1.05-19.24), HDL (OR = 4.19, 95% CI: 1.18-14.84), and TC (OR= 0.99, 95% CI: 0.46-2.09).

5.3 Gender Stratified Analysis

Table 5a demonstrates that for BMI, covariate associations including individual education, unemployment, and fast-food consumption differed between men and women. BMI was more positively associated to fast-food consumption in men; education was slightly and positively associated to BMI for men but strongly and negatively associated for women; and being unemployed was almost six times more positively associated with BMI in women than men. Furthermore, there were large gender differences in the associations between ALU and BMI, and ALU and TCR (Table 5b). These differences were substantiated statistically for TCR in a Model 4 interaction test that revealed an interaction RR of 1.39 (95% CI: 1.06-1.81). The beta coefficient for the interaction term in Model 4 for BMI was 1.99 (95% CI: 0.00-4.01).

5.4 Additional Associations

In addition to the associations observed for ALU in the BMI analysis, other associations were observed in Model 4, including considerable associations for individual unemployment and education, alcohol and fast-food consumption, a moderate association for physical activity and area-level education, and a negligible association for age. Individuals who were employed and individuals who had at least a high-school education had a significantly lower BMI compared to those who were unemployed or without high-school, respectively. Visiting a fast food restaurant one or more times in one week was associated with a higher BMI, whereas engaging in more physical activity in the week was moderately associated with a lower BMI. Living in an area that fell in the third category of the proportion of individuals with a grade nine education as their highest level of schooling was moderately associated with a lower BMI. Finally, heavy and moderate alcohol consumption was associated with a lower BMI when compared to those who abstain.

Important covariate associations in Model 4 for TCR included a minimal but highly significant association for physical activity, age, and gender. Living in an area that fell in the second or third category of the proportion of the population with grade 9 as their highest level of schooling was associated with a lower TCR score compared to the referent first category. Visiting a fast food restaurant more than once a week was associated with a slightly higher, but statistically significant risk. Having an income of greater than or equaled to $50,000 per annum was associated with a more favourable TCR profile. Finally, those who were classified as “heavy drinkers” had a lower cardiometabolic risk than abstainers.

6. MANUSCRIPT OF ARTICLE FOR SUBMISSION TO PEER-REVIEWED JOURNAL

Associations between area-level unemployment, body mass index, and risk factors for cardiovascular disease in an urban area

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Keywords: Neighbourhood; Unemployment; Cardiovascular Diseases; Residence Characteristics.

Counts: Abstract: 238 Text: 2982 Pages: 27 Tables: 3 Figures: 0

ABSTRACT

Introduction: Little is known about whether area-level unemployment is independently associated with individual-level Cardiovascular Disease (CVD) in an urban setting. Furthermore, it is unclear whether this relationship differs by sex. We sought to determine whether area-level unemployment (ALU) was associated with CVD risk, and whether this association differs by sex.

Methods: A sample of 342 individuals from the Montreal Neighbourhood Survey of Lifestyle and Health (MNSLH) self-reported behavioural and socioeconomic information. A registered nurse collected biochemical and anthropometric data. ALU was operationalised within a 250 m radius buffer centred on individual residence using a comprehensive Geographic Information System (GIS). Generalized Estimating Equations were used to determine if body mass index (BMI), and a cumulative score for total cardiometabolic risk (TCR) representing elevated values for total cholesterol, triglycerides, high-density lipoprotein cholesterol, and glycosylated hemoglobin, were associated with ALU.

Results: After adjustment for age, gender, smoking status, behavioural and socioeconomic covariates, living in an area in the 4^{th} and 3^{rd} ALU quartiles was associated with having elevated BMI (Q_4 beta = 2.1 kg/m^2 (95% CI: 1.02-3.20); Q_3 beta = 1.5 kg/m^2 (95% CI: 0.55-2.47) and greater TCR risk [Q_4 RR = 1.82 (95 % CI: 1.35-2.44); Q_3 RR = 1.66 (95% CI: 1.33-2.06)] relative to the 1^{st} quartile. Sex-by-ALU interaction revealed a 1.99 kg/m^2 (95% CI: 0.00-4.01) difference in BMI and 1.39-fold (95% CI: 1.06-1.81) greater TCR Score for women compared to men.

Conclusions: Area-level unemployment is associated with greater CVD risk in men and women but associations are stronger among women. **Words in Abstract: 250**

INTRODUCTION

Cardiovascular disease (CVD) has long been recognized as an important public health problem, [1, 2] and recent research has focused on how social and physical environments shape the distributions of its risk factors, [3-7] and outcomes. [8, 9] In this research, area-level social deprivation has garnered much attention. Often gauged by cumulative indices combining measures of education, income and occupation, there is mounting evidence that area-level social deprivation plays an important role in shaping population rates of CVD. [6, 9, 10] Furthermore, some studies have shown sex differences in how these environments are associated with the cardiovascular status of men and women. [11, 12]

Notwithstanding a focus on social deprivation, few studies have sought to evaluate the role played by individual components of common cumulative socioeconomic indices in the prevalence and incidence of CVD. Composite measures of area-level social deprivation are associated with CVD risk factors and events across the Western world. [4, 9, 10, 13-15] Yet the use of cumulative indices has detracted from tangible policy initiatives insofar as they conflate pathways and obscure the independent contributions of each component to specified health outcomes. [16] Only three studies have looked at whether ALU is, of its own, related to CVD risk factors or events. [15, 17, 18] This is surprising since ALU is a direct measure of urban deprivation. [19] By cutting across the gamut of social, political and economic "spaces," [20] ALU acts as an indicator of potential social polarization [21](p. 258) and social inequality, [22] and thus may reflect the differential distributions of CVD in a population. Evidence of such a relation would

support a tangible representation of social deprivation towards which policy initiatives could be directed.

The most common technique used to analyze area-health associations is to aggregate resident data to administrative group-levels for use in multilevel models. Yet there is a growing awareness of the limitations associated with arbitrarily defined administrative unit measures as ostensibly meaningful neighbourhood constructs. [23] Census tracts (CTs) and other administrative groupings do not correspond to residents' perceptions of their neighbourhoods, [24] and in contiguous urban areas residents who are closer in space are generally more alike than those farther apart. [25] Arbitrary boundaries that group residents into one or another unit impose distinctions that may not exist in reality. [26]

This study examined the associations between ALU and risk factors for cardiovascular disease in a field study of area-based characteristics and individual risk factors for cardiometabolic disease. To represent and ascribe neighbourhood influences, we used moving-window areas, which consider a perceptually relevant space around the individual, and improve problems of misclassification with those living on the margins of a given fixed-boundary. [26, 27] We hypothesized that ALU is associated with an elevated BMI and higher total cardiometabolic risk. Given known differences in the determinants of CVD in men and women, we explored whether any such associations would differ according to sex, accounting for behavioural, socioeconomic, and area-level covariates.

METHODS

Population and setting

Data for this study were obtained through the Montreal Neighbourhood Survey of Lifestyle and Health (MNSLH). The Island of Montreal, with a large urban centre and a diverse multi-ethnic population, provided the setting for our study. We sampled individuals within seven CTs representative of the distribution of CT level SES and language groups on the Island of Montreal using a stratified cluster sampling design. Six CTs were initially sampled, three primarily French speaking, the others primarily English speaking, across tertiles of an SES index combining educational attainment and income. An average of (n=49) individuals were randomly sampled within CTs. A seventh tract was added later to account for low participation in one CT.
Inclusion criteria were being between 18 and 55 years of age, without diagnosed cardiometabolic disease and able to read French or English. Informational material was left to all accessible non-commercial addresses within each CT, followed by a recruiter visit 48 to 72 hours later. A note was left to individuals absent at the first visit inviting them to contact research coordinators if they wished to participate. Up to four follow up attempts were made. Contact could not be established with residents of approximately 40% of addresses.

During the follow-up visit, participants were offered the opportunity to answer the questionnaire. Among those reached, approximately 80% were eligible, approximately fifteen percent of which agreed to participate. A total of 374 individuals completed the main questionnaire and were contacted for a visit. Three hundred forty-four participants provided biological data and two had missing age information, leaving us with a final sample size of 342 individuals. Participants for whom we lacked biological information

were mostly from French language households, but did not differ in gender, educational attainment, marital status, income, or fast-food consumption.

Questionnaires were completed on paper, by phone, or on the Internet. A registered nurse collected anthropometric measures and finger-prick blood samples during a home-visit. Point-of-care equipment (LDX cholesterol, and GDX hemoglobin A1c analyzers, Cholestech, Hayward, CA) was used to analyze blood samples on site. All participants gave their informed consent prior to participation. The study protocol was approved by the Human Research Ethics Committee of the Centre de recherche du Centre Hopitalier de l'Universite de Montreal (CR-CHUM).

Outcome Measures

Finger-prick blood samples obtained by the nurse were analyzed for glycosylated haemoglobin (% HbA_{1c}), triglycerides (TRG; mmol/L), total cholesterol (TC; mmol/L), and high-density lipoprotein cholesterol (HDL; mmol/L). Total cardiometabolic risk (TCR) was estimated as the sum of biological variables above clinical levels. Cut-offs were based on American Heart Association Guidelines for Primary Prevention of Cardiovascular Disease and Stroke: $HbA_1c \leq 7.0$ %; TRG ≤ 1.7 mmol/L; TC ≤ 5.0 mmol/L; HDL ≥ 1.29 mmol/L for women and 1.03 mmol/L for men. [28] Body Mass Index (BMI) was calculated as weight (kg)/height (m^2) and retained in continuous form. BMI and TCR were considered separately to ascertain whether area-level effects differentially influence anthropometric and haematological CVD antecedents.

Exposure measures and Covariates

Area Level Measures

Model covariates were selected using a Directed Acyclic Graph (details available on request from first author). Area-level information was derived from the 2001 Canadian Census incorporated into a comprehensive Geographic Information System. [29] The moving-window area [26] entailed creating individually-centred buffers with a 250 m radius to represent immediate "neighbourhood" influences, with census level data geo-linked to individuals' homes using GeoPinpoint© Software (DMTI Spatial).

For ALU, the census-based unemployment rate within a 250 m buffer zone centred on the individual's residence was calculated. A weighted average of the unemployment rate in each CT over which the buffer zone overlapped was calculated based on the overlap size. The same technique was used to operationalise area-level education as a proportion of the population 20 years and older with at least a grade 9 education. [30] ALU was categorized into quartiles. Area-level education was divided into three categories based on gaps in the variable's distribution.

Individual level measures

Physical activity was assessed via questionnaire inquiring about overall time spent walking, time spent walking *specifically* for health, and time spent in vigorous physical activity over the previous week. This information was converted to the number of Metabolic Equivalents (METS) expended over the previous week and operationalised as a standard score.

A modified version of the U.S. Behavioural Risk Factor Surveillance System questionnaire was used to assess fruit and vegetable consumption. [31, 32] Consumption of eight different groupings of fruits and vegetables over the previous week, ranging from "None" to "Every day," was documented. A total fruit and vegetable consumption

score was calculated based on the sum of responses to the eight five-point items and operationalised as a continuous variable.

Fast food consumption was estimated using the number of fast food restaurant (FFR) visits in the previous week as a proxy for consumption, assessed with a four-point scale from zero to 5 times or more per week. This score was dichotomized using a cut-off of one or more FFR visits in the previous week based on a split in the variable distribution. A score of zero was used as referent.

Smokers were categorized as smoker/non-smoker, with non-smoker as referent.

Alcohol consumption was measured via questionnaire on the quantity of alcohol consumed over the previous week. Responses were categorized as “abstainer,” “light drinker” and “heavy drinker” based on the 2005 USDA/HHS Dietary Guidelines. [33] Abstainer was used as the referent.

Individual-level socioeconomic covariates included education, income, and employment status. Education and income were assessed using two nine-point scales requiring respondents to indicate the highest level of education completed and total yearly household income, respectively. Education was operationalised as a dichotomous variable with greater than or equal to a high-school education as referent. Income was operationalised using two dummy variables for total yearly household income between $20,000 and $50,000 (0 = referent), and $50,000 plus (0 = referent).

Employment status was determined via questionnaire and operationalised as a dichotomous variable. Unemployed status was used as the referent.

Age was categorized as a continuous variable. Male was referent for the gender variable.

Statistical Analysis

Analyses were conducted using SPSS 14. [34] Generalized Estimating Equations (GEE) were used to simultaneously estimate the effects of area- and individual-level predictors on outcomes while accounting for clustering in CTs. [35-38] Associations with the continuous BMI measure were assessed using the identity link function and normal distribution, with results expressed as beta coefficients. A Poisson regression model was used to assess associations with TCR (a count measure), given as relative risks (RR). Census tracts were used as the clustering unit, with an exchangeable correlation matrix.

Four regression models were fitted to assess the relationships between ALU and outcomes. Covariates were introduced in blocks. Model 1 was the baseline model and included variables that fit criteria for a confounding variable. [39](p. 132) Model 2 included the Model 1 covariate block as well as area-level education. Model 3 included the two previous blocks and individual level socioeconomic variables. Finally, Model 4 adjusted for individual-level behavioural variables in addition to previous blocks. Model diagnostics included Pearson residuals plotted against the predicted value of the Linear Predictor. [40] Outlying values were identified visually based on their distance from the plot cluster and analyses re-run with outliers removed. No outliers were visible in the TCR model, four were identified in the BMI model. However, results did not differ between BMI models including and excluding outliers. Therefore all analyses were performed with complete data. Assessment of Variance Inflation Factors (VIFs) indicated no multicollinearity in the predictor variables.

RESULTS

Descriptive statistics

Behavioural, socioeconomic and biological characteristics of the study participants are presented according to sex in Table 1.

Table 1a. Sample characteristics of neighbourhood study participants (n=342).

	Men (n = 169)	Women (n= 173)
Continuous Variables	Mean (Std Dev)	Mean (Std Dev)
BMI (Kg/m^2)	25.07 (3.91)	24.60 (5.18)
Age (years)	35.77 (8.87)	33.89 (8.51)
Energy expenditure (Std Total Mets)	0.12 (1.05)	-0.17 (0.857)
Fruit & Vegetable Consumption (Max = 40)	13.15 (4.86)	14.23 (4.10)
Categorical Variables	N (%)	N (%)
Unemployed		
Yes	27 (16.0)	13 (7.5)
No	142 (84.0)	160 (92.5)
Area-Level Unemployment		
Quartile 4	33 (19.5)	43 (24.9)
Quartile 3	45 (26.6)	48 (27.7)
Quartile 2	47 (27.8)	48 (27.7)
Quartile 1	44 (26.0)	34 (19.7)
Fast Food Consumption		
Yes	87 (51.5)	61 (35.3)
No	82 (48.5)	112 (64.7)
Smoker		
Never smoker/former smoker	113 (66.9)	125 (72.3)
Smoker	56 (33.1)	48 (27.7)
Education		
Less than high school	9 (5.3)	18 (10.4)
High-School completed	35 (20.7)	26 (15.0)
Trade school or university	125 (74.0)	129 (74.6)
Alcohol Consumption		
Abstainer	55 (32.5)	64 (37.0)
Moderate	80 (47.3)	97 (56.1)
Heavy	33 (19.5)	11 (6.4)
Income		
Below $20K (CAD)	44 (26.0)	57 (32.9)
Between $20K & 50K (CAD)	61 (36.1)	52 (30.1)
Above $50K	64 (37.9)	64 (37.0)
Total Cardiovascular Risk		

0 no indicator exceeding risk value	39 (22.8)	62 (35.8)
1 indicator exceeding risk value	51 (29.8)	73 (42.2)
2 indicators exceeding risk value	44 (25.7)	28 (16.2)
3 indicators exceeding risk value	28 (16.4)	9 (5.2)
4 indicators exceeding risk value	7 (4.1)	1 (0.6)

In general, women had similar BMIs but a more favourable TCR profile relative to men. Relative to women, men exercised more, frequented fast food establishments and were unemployed more often, smoked more, and consumed more alcohol.

Associations between ALU, BMI and TCR

Relationships between ALU and BMI, and ALU and TCR are provided in Table 2 for statistical models 1 through 4.

Table 2. Association between area-level unemployment, body mass index (BMI) and total cardiometabolic risk (n=342).

		Model 1[b]		Model 2[c]		Model 3[d]		Model 4[e]	
		Parameter Estimates (Std Err)	p-value	Parameter Estimates (Std Err)	p-value	Parameter Estimates (Std Err)	p-value	Parameter Estimates (Std Err)	p-value
BMI	ALU4[a]	2.66 (0.15)	<0.001	3.20 (0.31)	<0.001	2.64 (0.17)	<0.001	1.89 (0.47)	<0.001
	ALU3	1.56 (0.20)	<0.001	2.05 (0.23)	<0.001	1.50 (0.30)	<0.001	1.32 (0.45)	0.003
	ALU2	.51 (0.17)	0.003	1.49 (0.54)	0.006	1.23 (0.36)	0.001	0.952 (0.64)	0.136
		RR (95 % CI)	p-value	RR (95 % CI)	p-value	RR (95 % CI)	p-value	RR (95 % CI)	p-value
TCR	ALU4[a]	1.61 (1.49-1.73)	<0.001	2.22 (1.58-3.13)	<0.001	1.91 (1.40-2.62)	<0.001	1.82 (1.35-2.44)	<0.001
	ALU3	1.47 (1.34-1.60)	<0.001	1.82 (1.42-2.34)	<0.001	1.57 (1.22-2.03)	0.001	1.66 (1.33-2.07)	<0.001
	ALU2	1.15 (1.07-1.24)	0.001	1.42 (0.99-2.02)	0.056	1.28 (0.93-1.77)	0.132	1.37 (0.97-1.94)	0.076

[a]Referent is first (lowest) quartile throughout
[b]Model 1 included age and gender
[c]Model 2 included age, gender, and area-level education
[d]Model 3 included age, gender, area-level education, and individual education, income and employment status.

[c]Model 4 included age, gender, smoking status, area-level education, individual education, income and employment status, physical activity, fast-food consumption, fruit and vegetable consumption and alcohol consumption.

A gradated relationship was apparent between ALU quartiles and both BMI and TCR. This relationship was unchanged after accounting for area- and individual-level covariates.

Body Mass Index

There was a monotonic and positive association between BMI and ALU. Relative to the first quartile, this relationship was statistically significant for quartiles 2-4 in models including age and area-level education. For quartiles 3-4 these associations were unchanged upon the inclusion of individual education, income and employment status (Model 3) and behavioural covariates (Model 4).

Total Cardiometabolic Risk

Similar to the BMI analysis, the association between TCR and ALU was significant and gradated after controlling for the sequence of covariate blocks. For quartiles 2-4, associations were unchanged upon the inclusion of age, area-level education, and markers of individual socioeconomic status when compared to the first referent quartile. Quartiles 3-4 remained significant after the addition of behavioural covariates into the model.

Covariate Associations

In Model 4, associations were also observed between BMI and area-level education, fast food consumption, individual education, alcohol consumption, physical activity and individual unemployment. These same variables were also associated with TCR, excluding individual unemployment and including individual income.

Gender Stratified Analysis

Gender specific models revealed differences in the magnitude of association for both BMI and TCR models (Table 3). These differences were substantiated statistically for TCR in a Model 4 interaction test (results not shown) that revealed an interaction RR of 1.39 (95% CI: 1.06-1.81). The beta coefficient for the interaction term in Model 4 for BMI was 1.99 (95% CI: 0.00-4.01)

Table 3. Association between area-level unemployment (ALU), body mass index (BMI) and total cardiometabolic risk (TCR) for 169 men and 173 women.

		BMI				TCR					
		Men		Women		Men			Women		
		beta (std err)	p-value	Beta (std err)	p-value	RR	Lower 95 % CI	Upper 95 % CI	RR	Lower 95 % CI	Upper 95 % CI
Model 1	ALU4	0.80 (0.24)	0.001	4.63 (0.35)	< 0.001	1.36	1.02	1.81	2.10	1.49	2.95
	ALU3	-0.32 (0.48)	0.514	3.65 (0.40)	<0.001	1.37	1.02	1.83	1.58	1.08	2.31
	ALU2	-1.70 (0.29)	< 0.001	2.53 (0.34)	<0.001	1.20	0.88	1.67	1.13	0.76	1.69
	ALU1	.	.	.	.	.	.	.	.	.	.
Model 2	ALU4	0.96 (0.98)	0.328	5.70 (1.91)	0.003	1.85	1.26	2.72	3.00	1.10	8.19
	ALU3	-0.53 (0.61)	0.932	4.50 (1.31)	0.001	1.56	1.16	2.11	2.09	0.83	5.25
	ALU2	-0.14 (0.96)	0.884	3.08 (1.08)	0.005	1.25	0.77	2.04	1.46	0.68	3.12
	ALU1	.	.	.	.	.	.	.	.	.	.
Model 3	ALU4	1.45 (1.16)	0.212	4.89 (2.07)	0.018	1.64	1.13	2.39	2.38	0.98	5.79
	ALU3	0.18 (0.70)	0.797	3.89 (1.34)	0.004	1.42	1.03	1.96	2.64	0.67	4.02
	ALU2	0.04 (0.93)	0.966	3.18 (1.18)	0.007	1.19	0.71	2.01	1.27	0.61	2.64
	ALU1	.	.	.	.	.	.	.	.	.	.
Model 4	ALU4	1.69 (1.10)	0.126	2.70 (2.11)	0.202	1.61	1.19	2.18	2.51	1.12	5.60
	ALU3	0.57 (0.70)	0.420	2.25 (1.69)	0.183	1.47	1.18	1.84	1.82	0.77	4.28
	ALU2	0.18 (1.21)	0.879	1.71 (1.57)	0.278	1.26	0.82	1.94	1.41	0.74	2.70
	ALU1	.	.	.	.	.	.	.	.	.	.

[a]Referent is first (lowest) quartile (ALU1) throughout
[b]Model 1 included age and smoking status
[c]Model 2 included age, smoking status, and area-level education
[d]Model 3 included age, smoking status, area-level education, and individual income, education and employment status.
[e]Model 4 included age, smoking status, area-level education, individual income, education and employment status, fresh fruit and vegetable consumption, fast food consumption, physical activity and alcohol consumption.

Discussion

In our study sample of urban residents spread across seven census tracts, we observed associations between area-level unemployment, BMI and cumulative risk for cardiometabolic disease. These associations held even when accounting for area-level education, individual-level education, income and unemployment status, fruit and vegetable, fast food, alcohol and tobacco consumption, and physical activity. There were marked gender differences in these associations, with women having stronger associations than men for relations between area-level unemployment and BMI, and a cumulative risk marker for cardiometabolic disease.

Our findings are consistent with two of the three studies that looked at area-level unemployment and the presence of CVD risk factors or the incidence of CHD. These studies, carried out in (i) a combined German and Czech, [17] and (ii) Swedish cohort, [18] documented relationships between area-level unemployment and obesity, [17] and first hospitalization for a fatal or nonfatal coronary heart disease event. [18] Neither study, however, took into account the role of behavioural variables in their measures of association, including smoking in Sundquist et al's study. In addition, Dragano et al looked only at individuals aged 45-69, thus omitting those who are most vulnerable to CVD events associated with BMI. [41]

The other study, based in Montreal, assessed the relation between BMI and community unemployment operationalised at the police district level in first and second-generation immigrants and non-immigrants. [15] The authors found no relation between community unemployment and BMI. However, BMI was based on self-reported height and weight, and operationalised as obese and non-obese. Categorical estimates of BMI

based on self-reported weight and height are prone to misclassification [42] that may lead to dependent error [39] which could partly explain why no association was observed.

Ours is the first study to demonstrate an association between area-level unemployment and cardiovascular risk in adults residing in a Canadian urban setting. Rather than focus on obesity, we used body mass index as a continuous variable, as well as an index of cardiometabolic risk that includes both lipid and glycaemic markers. We operationalised our neighbourhood variables based on moving window CT areas rather than larger administrative units, which may mask within unit variation of area-level unemployment. [43] Our sample included individuals aged 18-55 years, and we simultaneously accounted for behavioural, socioeconomic and another area-level SES indicator. Molinari et al [12] and Ellaway and Macintyre [44] have suggested that relationships between the social environment and health outcomes are likely to differ between men and women. In particular, Molinari et al [12] argue that women are more likely to be affected by the social environment of a given community compared to men, whereas men are more sensitive to variation in the physical community. We observed marked gender differences in the associations between area-level unemployment and both total cardiometabolic risk and BMI. Accounting for area-level education, and individual behaviour and socioeconomic status, we found a 1.4-fold greater risk in women compared to men in the TCR model, which represents the ratio of TCR risk from the 4^{th} to 1^{st} quartile in women, divided by the ratio of TCR risk from the 4^{th} to 1^{st} quartile in men. We also observed a risk difference of 1.99 kg/m^2 which represents the difference in BMI from the 4^{th} to 1^{st} quartile ALU in women, minus the difference in BMI from the

4th to 1st quartile in men. Whether these differences are due to (i) internal physiological or psychological differences; (ii) to external socially, economically, or politically produced differences; or (iii) some combination of the two, we cannot say.

Although we adjusted for a number of covariates, statistically significant associations persisted and were not "explained away" after we accounted for them. This persistence is difficult to explain given the extent of the influences that we accounted for. There most likely remained some unmeasured intermediate factors that play a role in channelling the effect of area-level unemployment to BMI and total cardiometabolic risk. One potential candidate is psychosocial status, measures of which are implicated as potential mediators of area-health relationships. [23, 45] Alternatively, we could speculate that part of this effect could be due to a direct link between the social environment and the individual, in which non-conscious stress perceptions influence the allostatic and cardiometabolic status of an individual. [23] Additional research is required to evaluate potential causal mechanisms through which area effects are expressed.

This study has a number of limitations. The cross-sectional design precludes causal inference. Our limited sample size translates into limited statistical power and impacts the precision of our point estimates. Self-selection of participants introduces potential bias, and the limited response rate would suggest that our sample might not be representative of the source population. However, out of approximately 20 Census sociodemographic measures, our sample did not differ from the source population, with the exception of age, marital and immigrant status, and education in 4 of 7 CTs. Age differences were expected, given our selection criteria of individuals being 18-55 yrs,

and it is known that individuals with higher levels of education tend to participate more willingly in epidemiological studies. [46] Our results are unlikely to reflect over controlling, since the nature of the associations evaluated remained consistent as new covariates were added to models. That we determined covariates using DAGs rather than more arbitrary criteria decreases the likelihood of introducing bias. [47] We did not account, however, for psychosocial factors or social networks—both of which may play a role in modulating the area-level-to-CVD relation. [48, 49]

Endogeneity [50] was not considered; our protocol did not ask whether residents resided where they did for health reasons. Also, we utilised a 250m buffer zone to represent immediate "neighbourhood" influences. The issue of scale has not yet been resolved in studies of area effects, and it is possible that other radii may be more or less appropriate. In summary, area-level unemployment in the proximal 250 m area to individual residence is associated with higher BMI and greater total cardiometabolic risk even after accounting for key area- and individual-level covariates. This association is greater for women than for men. The basis of this persistent association requires further investigation.

AKNOWLEDGEMENTS

The authors thank Ian Shrier MD, PhD for helpful assistance with conceptual aspects of the analyses, and Yan Kestens PhD for some helpful comments. We acknowledge Laurette Dubé, McGill University, principal investigator of a team grant providing partial support to the MNSLH.

COMPETING INTERESTS

None

FUNDING

Data collection was provided in equal parts through the (i) *Canada Research Chairs* program and the *Canada Foundation for Innovation* (grant #201252, MD), (ii) *Canadian Institutes of Health Research* (grant # 200203 MOP 57805, LG), and (iii) *Fonds de la Recherche en Santé du Québec* (FRSQ) (team grant # 8394, LD). At the time of this research, AIN was supported by a Canada Graduates Scholarship Master's Award from the Canadian Institutes of Health Research. MD was supported by a Canada Research Chair for Biopsychosocial Pathways in Population Health, awarded by the Canadian Institutes of Health Research. CP was supported by a postdoctoral fellowship from the Fonds de la Recherche en Santé du Québec. LG holds a Canadian Institute for Health Research / Centre de Recherche en Prevention de l'Obésité Applied Public Health Chair in Neighbourhoods, Lifestyle, and Healthy Body Weight. The funding sources did not participate in study design, data collection, analysis or interpretation, writing of the report, or in the decision to submit the paper for publication.

WHAT THIS PAPER ADDS

- A gradated relationship exists between area-level unemployment rates and cardiovascular disease antecedents, including BMI and a cumulative index for total cardiometabolic risk.
- This association persists after accounting for key individual-level socioeconomic status markers and behavioural practices, and an additional marker of area-level socioeconomic status.
- The novel contribution of this study is the observation that women had stronger associations than men in relations between area-level unemployment and

cardiovascular disease risk, even after accounting for behavioural, socioeconomic and other area-level covariates.

POLICY IMPLICATIONS

- Interventions to reduce cardiovascular disease risk and antecedent risk associated with BMI may benefit from an ecological approach addressing not only individual-level targets, but also on area-level socioeconomic influences including unemployment. Further, public health interventions might specific target areas characterized by high unemployment.
- Interventions aimed towards mitigating the impact of area-level unemployment on cardiovascular disease should consider the differential status this relationship holds relative to men and women.

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7. DISCUSSION

We set out to assess whether area-level unemployment, as a marker for area-level socioeconomic and physical characteristics, was related to the antecedents of cardiovascular disease—namely, BMI and a marker for total cardiovascular risk. Specifically, our aims were to examine whether area-level unemployment is associated with an elevated BMI and a higher total cardiovascular risk when considering the perceptually relevant spatial area centered on the individual's residence, and to determine if this relation holds after controlling for individually-based behavioural, SES, and area-level SES markers selected using Directed Acyclic Graphs. Furthermore, we investigated whether these relationships differ according to sex. To our knowledge, this is the first study assessing the relationship between area-level unemployment and antecedents to cardiovascular disease, while using a moving window area centered on the individual.

We found significant associations between quartiles of area-level unemployment centered on the individual and the antecedents of cardiovascular disease. Those individuals living in areas with higher unemployment rates were more likely to have higher BMIs and more unfavorable TCR scores than those who lived in areas with lower unemployment rates. Furthermore, although we did not carry out formal statistical analyses on the differences across quartiles to test for dose-response, we did observe a gradated relationship across quartiles. This relationship held even after the inclusion of individually based area-level education, and individual behavioural and socioeconomic covariates.

Our findings, as with most of the findings on social deprivation and health, point towards the existence of an association between area-level unemployment and cardiovascular risk. Furthermore, in conjunction with the few studies that have looked at gender differences, there seems to be a large gap in the observed associations, with women being at higher risk than men living in an area with the same unemployment rate. All in all, this research supports the notion that socially deprived neighbourhood areas engender deviations in the cardiovascular and metabolic function of individuals living in those areas.

Studies on the relationship between urban social deprivation and health tend to use markers of poverty, income inequality, and combined indices of employment, income and education such as socioeconomic position (Oakes & Kaufman 2006). Despite the increasing interest in the area-level deprivation-health relationship, only a small body of public health literature exists using area-level unemployment rates as a marker for neighbourhood deprivation. Yet the Urban Geography and Demography literature documents numerous instances of unemployment rates as indicators of area-level social and economic deprivation. According to Langlois and Kitchen (2001) the bulk of urban and social deprivation rests upon economic factors. Elsewhere, Kitchen (2001)[(p. 1907)], based on the results of a Principal Component Analysis and supporting literature, justifies the use of unemployment as a "direct measure of urban deprivation." Pacione (1995) contends that the principal cause of deprivation is economic and rests on: (1) unemployment; and (2) low wages earned by those in declining industries. Finally, Wilson has argued on numerous occasions (1987; 1993; 1996) that much of the

social deprivation experienced by individuals and communities is largely attributable unemployment and the disappearance of work.

Furthermore, the few public health studies that have looked at the ALU-health relation support the findings in Urban Geography. Bosma et al (2001) found an association between the percent of individuals in a neighbourhood who reported that they were unemployed or disabled that was independently related to mortality. Curtis et al (2004) found that, of all the variables assessed in their study on neighbourhood conditions in the 1930s and individual health in the 1990s, only the unemployment rate in the 1930s was related to the individual's health status in the 1990s. After limiting their gender based analysis to the employed sub-sample of their study, Stafford et al found that gender-based differences in health disappeared in all measures except for ALU (Stafford et al 2005). Finally, Cummins et al (2005a) found that ALU was associated with fair to very bad self-rated health independent of individual age, sex, social class and economic activity. All of these studies that found important associations for ALU did so in a European context, which presents itself with unique socioeconomic and cultural conditions compared to Canadian cities. For one, unemployment may not be as big a problem in Montreal as it is in European cities. Therefore, if it can be established that ALU is an important issue in Montreal, an assessment of the role of ALU in relation to CVD becomes all the more warranted.

7.1 Social Disadvantage in Montreal

The unique socioeconomic circumstances of the city of Montreal, with a total population of approximately 1,854,442 as of the 2006 census (Ville de Montréal 2008), have long been known. Until at least the late 1970s, Montreal's economic structure

could be classified as typically Fordist, implying a close relationship between its residential and economic structures (Lewis 2000). The relationship between Montreal's economic structures, the shape of residential communities, and the population's health in Montreal has been known for over a century. As indicated in Ames' 1897 treatise, higher rates of infection, disease and mortality occurred in those living "below the hill," in the concentrated populations of Irish and French-Canadian unskilled workers in Griffintown and Pointe St. Charles, South of the wealthier Westmount area. The populations who inhabited the poorer areas below the wealthier Westmount area were often among those most vulnerable to the economic vagaries of the day (Ames 1972; Copp 1974).

As in the mid to late 19th century, contemporary economic transitions still affect the most vulnerable of Montreal's population. Kitchen (2001) found that more than half of the neighbourhoods in Montreal-East—an already disadvantaged sector—experienced increasing socioeconomic deprivation, with increases observed in male, female and youth unemployment, in lone female-parent families, in poverty, and in low-income families between 1986 and 1996. In fact, the spatial concentration of poverty is the highest in Montreal compared to all other major Canadian metropolitan areas (Seguin & Divay 2002, cited in OECD 2004)(p. 74).

The means by which these changes occur are many, but include primarily an out-migration of the middle-class residents to the suburbs and subsequent erosion of the tax-base in boroughs such as Montreal-North and Saint-Leonard (Marois 1998). This coincides with a significant gentrification of other boroughs such as the Plateau Mont-Royal, Old Montreal and Petit-Bourgogne (Little Burgundy) resulting in an exacerbation

of the gap between the have and have-not boroughs (Ley 1996). Furthermore, the uncertainty of Quebec's future as a part of Canada has created unique social and economic conditions in Montreal, compared to other Canadian cities, which, according to some authors, have accentuated the economic pressures faced by Montrealers (Alesina et al 2000; McCallum 1992).

Indeed, with respect to economic indicators, Langlois and Kitchen show that Montreal stands as "the most distressed city on the list" of ten major cities in Canada, including Toronto, Vancouver, Quebec City, and Winnipeg (Langlois & Kitchen 2001)[(p. 125)]. This finding is confirmed by a report from the Organization for Economic Cooperation and Development (OECD 2004) pointing out that, in Montreal, ever since the political uncertainties and economic crises of the 1990s, unemployment rates remain the highest (8.4 % in 2002) among the largest Canadian cities (7.4 % in Toronto and 7.8 % in Vancouver). Again, as Kitchen (2001)[(p. 1902)] points out, in 1996 Montreal had "one of highest rates of unemployment among Canadian metropolitan areas, one of the lowest median family incomes, and the largest share of low-income families (representing more than one third of all families in the central city in 1996)", once more invoking the economic recessions of the early 1990s as the likely culprit behind these inauspicious figures.

This brief assessment of the history of social deprivation in Montreal renders our analysis of the association between area-level unemployment and cardiovascular disease particularly germane to a broader public health perspective. Rather than being a moot issue, unemployment has been a dominant and recalcitrant feature of Montreal's socioeconomic scene. But limiting the discussion solely to how our operationalised area

unemployment variable stands in relation to cardiovascular health would not do justice to the complexity of the issue. Rather, in order to draw out inferences relevant to the larger scheme of things, a contextualized discussion of ALU is in order.

7.2 Area-level Unemployment in Context

The bulk of contemporary research into the social determinants of health has been occupied with drawing inferences based on variables of social deprivation operationalised for the purpose of regression modeling. At a deeper level though—and despite the fact that measures of inequality and other proxies for area-level deprivation have been used throughout the epidemiological literature—there is a lack of consensus regarding the theoretical status that these measures hold with respect to underlying societal types. There are those who view inequalities as a primary determinant of social types. That is, greater relative differences in a given society leads to the creation of highly differentiated social hierarchies and the loss of social cohesion which, in turn, result in negative psychological, neurological, and biological experiences that increase allostatic loads and, subsequently, results in the development of disease (Coburn 2004; Kawachi et al 1997; Kawachi et al 1999; Wilkinson 1997).

Others, however, regard inequality as a consequence of embedded social types and patterns, rather than as a cause. The notion that social determinants of health, such as income inequality, are somehow "embedded" into a larger context has proven to be quite difficult to manage in the context of traditional epidemiological studies—the most revealing example of which deals with the way social variables are treated in epidemiology. For example, McQueen and Siegrist point out that "[t]he most telling weakness of traditional epidemiological research into the role of social factors in disease

is the oversimplification of social variables." (McQueen & Siegrist 1982)[p. 353]. They lament the fact that "many epidemiological studies treat social variables as if they were biological variables and simplify them to the point where their meaning is questionable."[p. 353]. Muntaner (1999) points out that "social epidemiology has not provided better explanations [because of] a lack of *social* theory development, due mainly to the reluctance of epidemiologists to think about social mechanisms." (emphasis in original)[p. 121]. By looking at their social, political and historical contexts, inequalities can be seen as consequences of "fundamental changes in class structure which have produced not only income inequality but also numerous other forms of health-relevant social inequalities." (Coburn 2004)[p. 43].

Among the other forms of health-relevant social inequalities exacerbated by these "fundamental changes" stands unemployment. In fact, the institution of those same policies that occurred in the early 1980s, and that preceded the advent of rising income inequality around the world, also saw rises in unemployment on a global scale. The political transitions that resulted in economic policy shifts from Keynesian "state interventionism" to "laissez-faire capitalism" produced a growth in unemployment in all OECD countries (Table 6a & 6b), an increase in social inequalities, and a decline in social expenditures (Glyn 1995; Navarro 1998).

According to some authors, the economic analysis of these increases has proven inaccurate and the conclusions based thereon untenable, especially from a public health perspective. For example, it was supposed that the high inflation rates of the late 1960s and early 1970s led to a large difference between the nominal and real price of petroleum that subsequently triggered a global economic recession, culminating in

massive worldwide unemployment (Gilpin 2001). Yet Navarro (1998) questions the validity of any putative relationship between the 1973 oil crisis and rising unemployment, citing increasing rates of unemployment throughout the 80s and 90s, despite the lack of similar economic circumstances. Furthermore, the fact that the critical shortage in raw materials, oil price increases, and other economic shocks brought about by the 1950 War in Korea did not reproduce the same surges in unemployment undermines such a contention. Rather, according to Navarro (1998)[(p. 629)] these surges can be seen as the result of more fundamental causes, including the "political unwillingness and inability to regulate international financial markets…" that characterize the international political and economic ideology of the day.

The notion that social determinants of health are influenced mostly by issues of international policy and legislation (which, incidentally, are based primarily on human agency and choice rather than biological necessity) is corroborated in the literature outside of Public Health and Epidemiology as well. Gilpin (2001)[(p. 60)] confirms the lack of relevance and coherence in the creation and implementation national policies mentioned by Navarro, and adds that they have heavy and negative international consequences. Pointing to discordant political moves between the U.S. and Western Europe that "contributed to the instabilities in the world economy throughout the 1970s," and that made unemployment an international problem, he goes on to criticize economic policy makers for ignoring "crucial aspects of social reality that cannot be modeled or made consistent with neoclassical assumptions." (Gilpin 2001)[(p. 70)].

From a public health perspective, the crux of the issue is that many of the social determinants of health being assessed today, and particularly unemployment, are in

some way reflective of policy, and responsive to events; social, political and economic, both foreign and domestic (Coburn 2000; Gilpin 2001; Navarro & Shi 2001). These contexts need to be addressed, whether or not they can be included in statistical models. Markers such as area-level unemployment, with roots in economic, geographic, political and social phenomena, can be viewed not only as disconnected consequences of juridical, political, and economic decisions made at the local level, but should be considered as dialectically evolving phenomena responding to local, national, and international economic norms, cultural values, and policy changes.

7.3 MONTREAL IN THE GLOBAL SCENE

This perspective becomes all the more germane to our analysis when considering a cosmopolitan metropolis such as Montreal, which stands out on the global scene as it adapts to changing international political and economic circumstances. According to an OECD report (OECD 2004)[(p. 13)] international changes such as the implementation of the North American Free Trade Agreement (NAFTA) have allowed Montreal to increase its prominence in the international market as well as in Canada. In the global market, Montreal is known to be strong in the aersospace, biotechnology, culture and fashion industries, and Information Technology. Yet, as it stands, the report classifies Montreal unfavourably as a "metropolis in transition," (Figure 5) placing well behind many economically comparable cities across the globe. According to the OECD, Montreal ranks 44th out of a selection of 65 comparable OECD metropolitan regions with regards to real GDP per capita for 2001 (OECD 2004). This ranking has led the OECD to recommend that Montreal implement policies to "increase productivity, [and] reinforce existing regional clusters … that support innovation and attract high-skilled talents."

(OECD 2006)[p 25].

From an economic perspective the virtues of supporting innovation and attracting high-skilled talents are readily acknowledged. However, the influence of international economic competition on domestic autonomy—which, in a profit-motivated clime, undermines the ability of those autonomous political units to deal with degrading social conditions—is becoming a topic of analysis in many areas of academia. Robert Gilpin, Professor Emeritus at the Woodrow Wilson School of Public and International Affairs, Princeton University, refers to the demands of "integration … into the world economy, the intensifying pressures of foreign competition, and the necessity to be efficient in order to survive economically…" as prime forces that lead to the development of new forms of social organization, among which is ever-increasing inequality (Gilpin 2001)[p. 81].

Indeed, the current economic milieu, rather than "lifting all boats," seems to have disproportionately favoured the well-off (Haines 2001) while undercutting the capabilities of the those in need (Sen 2000). And given that "the single most important feature of economic development is that it is *uneven*," (Knox et al 2004, emphasis in original)[p. 287] a public health approach should not be so inclined to sanction a narrow economic focus as outlined by the OECD. To be fair, although the OECD has acknowledged that something needs to be done about the problems of social deprivation and income inequality, the impact that these acknowledgements have on their policy recommendations are minimal (David et al 1998). The ever-burgeoning increases in inequality noted after the implementation of similar market oriented policies, and the subsequent health disparities thought to arise there from should serve as a cautionary

example against such de-contextualized policy reasoning, especially if the focus of such policies is to "reinforce existing regional clusters" at the expense of already deprived areas. If our observed association between area deprivation and cardiovascular disease proves causal, then the implications of uneven economic development on the Island of Montreal would have health-related implications that need to be considered.

7.4 CURRENT FINDINGS

Previous studies that have looked at the relationship between area-level social deprivation and cardiovascular disease have been limited by a number of important constraints. Firstly, as highlighted by Diez-Roux et al (2001)[p. 103] numerous studies that have found a geographic patterning of CHD due to area-based deprivation have been unable to implicate the variation of individuals as involved in those area-based differences (Elford et al 1989; Fabsitz & Feinleib 1980; Wing et al 1988; Wing et al 1987). Secondly, those studies that have linked area-level indicators of social deprivation to the individual have tended to use arbitrarily defined pre-existing definitions of 'area' rather than a definition with more merit relative to individual circumstances.

In our study, we related area-level unemployment (or unemployment rates) to the individual using Generalized Estimating Equations (GEE) in order to correct for clustering at the CT level. Furthermore, we chose to operationalise our area based on a moving-window concept, allowing for a more theoretically relevant conceptualization of the surrounding "neighbourhood." We found that there exists an association between area-level markers of social deprivation, such as area-level unemployment, and markers for cardiovascular disease, such as BMI and an index for cardiometabolic risk. Using

our GEE models, we found that this association holds even when accounting for other markers of individually-based area-level socioeconomic status, such as area-level education; as well as when accounting for individual-level socioeconomic factors such as education, income and unemployment status; and individual-level behavioural variables, such as fruit and vegetable, fast-food, alcohol and tobacco consumption, and physical activity.

A gradated relationship was observed for all quartiles of area-level unemployment in both models accounting for all four covariate blocks. For BMI, individuals living in an area in which the unemployment rate fell in the fourth (or highest) quartile was associated with a 2.7 Kg/m^2 greater BMI when compared to the first (or lowest) quartile ($p < 0.001$). This relationship to the first quartile tapered off in magnitude when assessing the 3^{rd} and 2^{nd} quartiles to 1.6 and 0.5 Kg/m^2, respectively. This gradated relationship held, even when accounting for other area-level covariates (Model 2); when accounting for other area-level covariates *and* individual level socioeconomic covariates (Model 3); and finally, when accounting for other area-level covariates, individual-level socioeconomic covariates, *and* individual-level behavioural covariates (Table 3). In the model that contained all covariates, living in an area that fell in the fourth, third, and second area-level unemployment quartiles was associated with a BMI in excess of 1.9 Kg/m^2, 1.4 Kg/m^2, and 0.9 Kg/m^2 respectively, when compared to the first quartile.

The same trends were observed when we assessed the magnitude of total cardiometabolic risk, measured as a summated score of established markers for cardiovascular and metabolic disease, in relation to area-level unemployment. Once

again, a gradated relationship was observed. In the model that accounted only for well defined confounders, we found that living in an area that fell in the fourth, third and second quartile unemployment rates was associated with a 1.6-fold, 1.5-fold and 1.2-fold relative risk, respectively, when compared to the first quartile. This time, accounting for a gamut of area- and individual-level confounders increased the magnitudes of the relative risks, but did not change the observed dose-response trend.

We also found relationships between a number of covariates and cardiovascular status, one of which merits particular attention. The consumption of excess alcohol was associated with a healthier cardiovascular status as per our outcome measurements than those who abstained. From a certain point of view, this can seem peculiar given that ethanol and fats are very alike from a nutritional perspective (7 Calories per gram in ethanol versus 9 Calories per gram in lipids), while ethanol itself offers little more in nutritive value relative to lipids. However, in our questionnaire, we did not discern the type of alcohol consumed, and, although debated in the literature, there is evidence pointing to a cardioprotective effect of red wine (Saremi & Arora 2008). Furthermore, recent work by Fillmore et al (2007) has outlined the importance of the misclassification of abstainers who chose to abstain due to some pre-existing cardiovascular condition. Given that an understanding of the role of alcohol consumption in cardiovascular risk was not a part of our objectives, we did not take steps towards the remediation of such threats to the validity of our alcohol consumption variable.

7.5 Consistency of Study Results with Existing Literature

Our results are consistent with two of the three studies that looked at area-level unemployment and the presence of cardiovascular risk factors or the incidence of

coronary heart disease so far. These studies, carried out in (i) a combined German and Czech cohort (Dragano et al 2007), and (ii) in Sweden (Sundquist et al 2006), have documented relationships between area-level unemployment and obesity (Dragano et al 2007), and first hospitalization for a fatal or nonfatal coronary heart disease event (Sundquist et al 2006). Neither study, however, took into account the role of behavioural variables in their measures of association, including smoking in Sundquist et al's study. In addition, Dragano et al looked only at individuals aged 45-69, thereby omitting those who are most vulnerable to CVD events associated with BMI (Stevens et al 1998).

The other study, based in Montreal, assessed the relation between BMI and community unemployment operationalised at the police district level in first and second-generation immigrants and non-immigrants (Zunzunegui et al 2006). Using hierarchical linear models, the authors found no relation between community unemployment and BMI. However, BMI was based on self-reported height and weight, and operationalised as obese and non-obese. Categorical estimates of BMI based on self-reported weight and height are prone to misclassification (Rowland 1990) which may have led to a dependent error (Rothman et al 2008) that could partly explain why no association was observed.

Ours is the first study to demonstrate an association between area-level unemployment and cardiovascular risk in adults residing in a Canadian urban setting. Rather than focus on obesity, we used body mass index as a continuous variable, as well as an index of cardiometabolic risk that includes both lipid and glycaemic markers. We operationalised our neighbourhood variables based on a moving window census tract areas rather than larger administrative units, which may mask within unit variation of

area-level unemployment (Stafford et al 2008). Our sample included individuals aged 18-55 years, and we simultaneously accounted for behavioural, socioeconomic and another area-level indicator of socioeconomic status.

The findings outlined here are fall in line with a number of studies looking at social deprivation in general, and cardiovascular health. For instance, a study in Scotland (Davey-Smith et al 1998b) found that cardiovascular disease risk factors and cardiovascular disease specific mortality were associated with area-level deprivation represented as a composite measure of male unemployment, overcrowding and car ownership. They found relations between blood cholesterol concentration, angina, and smoking and area deprivation in both men and women, and between BMI, ECG ischemia and area deprivation in women alone. These associations remained after accounting for individual social-class, with the exception of cholesterol. Studies in the US (Diez-Roux et al 2001; Diez-Roux et al 1997; Wang et al 2007) and Canada (Ross et al 2007) have also documented similar patters between incident coronary heart disease (Diez-Roux et al 2001; Diez-Roux et al 1997) or BMI (Ross et al 2007; Wang et al 2007) and neighbourhood exposure variables incorporating measures of occupation, education, and income. All of these studies found a higher risk between area-level deprivation and coronary heart disease or cardiovascular risk markers.

Molinari et al (1998) and Ellaway and Macintyre (2001) have suggested that relationships between social and physical environmental factors and health outcomes differ in men and women. We observed gender differences in the associations between area-level unemployment and total cardiovascular risk. Accounting for area-level education, and individual behaviour and socioeconomic status, we found a 1.4-fold

greater risk in women compared to men in the TCR model, which represents the ratio of TCR risk from the 4th to 1st quartile in women, divided by the ratio of TCR risk from the 4th to 1st quartile in men. We also observed a risk difference of 1.99 kg/m^2 which represents the difference in BMI from the 4th to 1st quartile ALU in women, minus the difference in BMI from the 4th to 1st quartile in men.

Gender differences in the magnitudes of association for socioeconomic versus behavioural variables were also observed, and statistically tested. Stronger associations were observed for fast-food consumption and physical activity in males, and education and employment status in females. These results fall in line with previous empirical studies that found stronger associations in women for social and economic characteristics associated with self-rated health (Stafford et al 2005), and coronary heart disease classified as ICD codes 9 and 10 (Sundquist et al 2006), suggesting that, indeed, men and women respond differently to social and physical environments. However, whether these differences are due to: (i) internal physiological or psychological differences; (ii) to external socially, economically, or politically produced differences; or (iii) some combination of the two, we cannot say. Future research could look at whether gender-based differences in socioeconomic, physical, psychological, and/or cultural competencies and resources can explain gender-based differences in CVD risk observed here and elsewhere.

In considering the debate about whether or not to include intermediate variables in statistical models assessing relationships between area-level factors and health, we decided to do both. We used four different statistical models to account for a number of covariates and intermediates in the relationships between area-level unemployment and

BMI and cardiometabolic risk. Justifying selected covariates based on the use of directed acyclic graphs (DAG) for each model set, we ran our first and second models with well-defined confounders. The third and fourth models were run with covariates that are traditionally included as confounders in area-level research, but that may be judged as intermediates in the causal pathway.

Including an alternative marker of area-level socioeconomic status as a covariate in our model increased the association between area-level unemployment and both BMI and total cardiometabolic risk. Addition of individual-level income, education, and employment status slightly lowered the observed associations in both models, and the inclusion of behavioural covariates lowered the associations further, except in quartiles 2 and 3 of the total cardiometabolic risk model. Specifically, we saw an 18% increase in the quartile 4 beta for area-level unemployment in the BMI model after including area-level education, and a subsequent 15% and 22% decrease after adding individual-level socioeconomic and behavioural variables, respectively.

Despite the large number of covariates/intermediates in our models, we still observed statistical associations between area-level unemployment and both CVD antecedents. This is possibly due to some unmeasured intermediate variable that plays a role channeling the effect of area-level unemployment to BMI or total cardiometabolic risk. One potential candidate includes psychosocial variables, which are known to be potential mediators of area-health relationship (Marmot et al 1998). Alternatively, we could speculate that part of this effect is due to a direct link between area-level unemployment and the individual, in which non-conscious area-level stress perceptions alter the cardiometabolic and allostatic status of an individual (Daniel et al 2008).

More broadly, however, the mechanisms that can possibly explain our findings can be divided into two general categories: (i) physical, which can be further divided into direct and indirect mechanisms (Daniel et al 2008); and (ii) intersubjective. Physical causes that act through indirect mechanisms are defined as those resource barriers that are derived from, and inherent in, the structure of the built environment. These include the lack of nutritious or abundance unhealthy food choices (Morland et al 2002); the lack of opportunity to engage in meaningful physical activity, such as limited walkability (Gauvin et al 2005) or the lack of recreational parks and facilities (Papas et al 2007). Physical causes acting through direct mechanisms are the result of environmental stressors such as noise or various pollutants that can directly and non-consciously elevate allostatic loads and lead to cardiometabolic shifts which predispose individuals to weight gain or dyslipidemia by influencing catecholamine, insulin, lipid, oxidative and/or inflammatory processes in the individual exposed to such stressors.

Non-physical causes can also influence health outcomes in populations. Recent studies looking at how the relations between individuals are associated with cardiovascular risk point to other possible mechanisms independent of physical or environmental context (Christakis & Fowler 2007; 2008). They depend, rather, on intersubjective norms—or norms which are embedded in social practices (Abizadeh 2001)$^{(p.\ 27)}$—that continually reproduce the phenomena related to CVD risk. From an academic perspective, the idea of intersubjectivity provides a critical foundation on which epidemiologists can frame societal (as opposed to individual) health problems. For example, Charles Taylor, referring to intersubjective meanings notes that

> It is not just that people in our society all or mostly have a given set of ideas in their heads and subscribe to a given set of goals. The meanings and norms implicit in these practices are not just in the minds of the actors but are there in the practices themselves. (Taylor 1985)[p. 36]

It follows from such a line of reasoning that individually oriented health policies aimed at changing behaviour can only do so much. These interventions do not address the intersubjective aspects of illness or disease at the "level of shared norms and principles embedded in social and state practices and institutions" (Abizadeh 2001)[p. 32] that play an important part in framing and perpetuating patterns of disease.

With respect to our study, it is likely that both physical and intersubjective mechanisms played a role in the relationship between area-level unemployment rates to elevated BMI or TCR risk. For example, the compositional nature of ALU—that is, the fact that it is based on individuals in a given society—opens up the possibility of the existence of intersubjective social norms coterminous with ALU that predispose individuals to CVD risk. Such intersubjective practices can be buttressed by economic ideologies that result in the differential allocation of *physical* resources based on the characteristics of a given area. For example, areas with lower housing market values are more likely to attract individuals with limited economic resources, such as the unemployed. This concentration of unemployed individuals eventually leads to higher ALU. Lower market and real-estate values are also likely to attract unhealthful business establishments and repel healthful ones (Block et al 2004; Cummins et al 2005b), creating an overall physical environment that is not conducive to developing or maintaining healthy patterns of behaviour (Lynch et al 1997).

7.6 Discussion Synopsis and Public Health Implications

The logic of the argument put forth in the discussion can be summarized briefly as follows: Our empirical findings indicate that an association exists between area-level unemployment and CVD antecedents. Area-level unemployment is a common marker of social deprivation in Urban Geography and Urban Sociology, making it a suitable candidate for measuring the effects of social deprivation on health in epidemiology. Epidemiological studies have found associations between area-level unemployment and health, but only a few have looked at cardiovascular disease. The historical record indicates that Montreal has had issues with unemployment and socioeconomic development since at least the late 19th century. These issues have continued up until this day, increasing the likelihood that if any relation exists between area-level unemployment and health, it will be of great import in Montreal.

Yet the idea that area-level unemployment, as it is operationalised in our regression model, somehow shapes the health of the population of its own is unlikely. To look at area-level unemployment out of the context of its international social, political and cultural settings would do little more than undercut the abilities of policy makers to deal with the problems that arise from increasing local rates of unemployment. Understanding how area-level unemployment is intricately tied into the international social, political and economic fabric is a first step towards remedying the health problems that arise there from. This is especially true for a city like Montreal, whose prominence in the international scene renders it vulnerable to downturns in foreign economic development, and inappropriate foreign policies. Finally, the idea that broad-scale international factors shape the social and physical localities that eventually alter individual health status needs to be coupled with the notion of intersubjective

norms between individuals that reinforce the negative impact of area-level effects on health. These two concepts provide a rich framework on which we can base policies directed not only at individuals, but towards those broader societal forces that shape the health of populations.

From this discussion, one can clearly see how the public health implications of these findings involve both praxis-based and epistemological dimensions. From a praxis-based perspective, actions directed towards altering the distribution and frequency of cardiovascular disease in the population need to consider the interactions between local economic policy and urban form, and the influence of international economic pressures, ideals, and goals on local economic policy, that tend to reinforce competitive regional clusters at the expense of deprived ones. Public health policy makers should be informed about those economic and business practices that accentuate existing disparities for the sake of gaining a more stable market foothold, and be involved in finding ways to encourage economic development that will not detract from the population's health. This also implies that public health policy makers need to challenge fragmented approaches to policy making, and make connections between economic and public health policy silos such that the policies made in other areas will not detract from or annul those efforts made in the public health policy arena.

From an epistemological perspective, the findings and discussion outlined in the previous pages imply that the science of public health needs to orient itself towards a direction that is not solely engaged with individual-level phenomena. If public health is what we do, as a society, to ensure the health of the population (Institute of Medicine, 1988) then an understanding of the relationships between society-wide structures and

institutions, and the distribution of health and disease in a population is paramount. This implies that the science of public health needs to focus on the context in which disease unfolds. How does this context (social, political, economic and other) influence the dynamics of population-level disease incidence? How is this context influenced by international and transnational trends? These questions entail the utilization of frameworks that incorporate notions of hierarchy (Ahl and Allen, 1996), that take a top-down and bottom-up perspectives (Hutchins, 1996), and that look to trans- and inter-disciplinary frameworks for understanding the complex interactions between context and health.

8. LIMITATIONS

This study has a number of limitations. One of the first, and perhaps most obvious, of these results from the need to simplify our examination for analytical reasons. Referring back to Figure 2 of our study, we note the numerous phenomena, both "upstream" and "downstream" to ALU that merit serious investigation. In particular we did not assess how individual psychosocial characteristics, such as one's ability to manage stressful situations, plays in when faced with the situation of living in a high ALU region. Future research may reveal that those who posses more capable mastery skills are able to mitigate the detrimental effects of living in a high-ALU environment. Furthermore, investigation into the individual's home or life situation may highlight the importance of individual living circumstances in the relation between ALU and CVD risk, and may even provide an explanation as to why we found such drastic gender differences in our sample.

Another limitation is found in the operationalisation of some of our variables. Notwithstanding the need to contextualize our operational ALU variable, we also found that in both models, visiting a fast-food outlet more than once in the last week is associated with an elevated BMI and TCR. But since we did not assess the actual consumption of fast food, we cannot say definitively whether this means that the consumption of fast food is associated with BMI and TCR. The possibility that our marker for fast-food consumption represents an overall tendency toward unhealthful behaviour, rather than a specific marker of fast food consumption, cannot be excluded. In order to disclose whether fast food consumption is associated with BMI and TCR, one would need to measure the type and quantity of fast food consumed with more precision than done here. Yet given that this was not our aim, we settled for a measure of frequenting fast food establishments.

8.1 Inferential Limitations

In order to fully appreciate the implications of our findings, a number of other key limitations need to be systematically highlighted. First, due to the cross-sectional nature of our study design, the inferences drawn there from are confined to being to associative, rather than causal. Classical criteria for defining a factor as causal in the biomedical sciences, first outlined by Austin Bradford Hill in 1965, include: (1) the strength of the association; (2) consistency of the findings in other settings and scenarios; (3) specificity of the relationship between a putative cause and single effect; (4) temporality, or the antecedence of a putative cause to effect; (5) existence of a biological gradient or dose-response relationship; (6) the coherence of the association with existing theory and knowledge; (7) existence of a plausible rational for the causal

relation; (8) manipulability in an experimental setting; and [although omitted from some modern renditions of Hill's causal criteria (Last 2001)] (9) analogical reasoning, or, the inference of a putative effect based on similar previously experienced scenarios (Hill 1965). It is important to mention that, although Hill did not intend his nine points to become a "causal checklist" by which the presence or absence of causality can be definitively revealed (Phillips & Goodman 2004), these points do provide us with a framework that can be used to support reasoning with causal inference, or "scientific common sense" (Phillips & Goodman 2006).

The cross-sectional nature of our study design precluded us from obtaining information on criterion number 4, or the temporal relationship between our exposure and outcome. Some authors regard this criterion to be among the most important of criteria, one of the three "absolute requirements" needed in determining causality (Kaufman & Poole 2000(p. 108); Susser 1991(p. 638)). An additional criterion not discussed in Hill's 1965 publication, but which is related to the temporal criterion is that of directionality. In effect, directionality hinges on the fact that a change in an outcome is a consequence of the change in the determinant under investigation (Susser 1991)[(p. 639)]. Again, the cross-sectional nature of our study precludes such an analysis.

We were also unable to demonstrate manipulability of our exposure-effect relation, due to the observational nature of our study. However, it is worth mentioning that the majority of epidemiological studies, and especially those in Social Epidemiology, are limited by such an approach. There is a large and growing body of literature on the limitations of observational analysis (Rosenbaum 1984; 1995; Rubin 1978; 1990), especially in social epidemiology (Cooper & Kaufman 1999; Kaufman &

Cooper 1999; Muntaner 1999). Suffice it to say that given the nature of the current project, and the complexities associated with the limitations of observational studies, we chose not to address this issue.

8.2 INTERNAL VALIDITY

Rothman et al (2008)[p. 129] outline three general categories that comprise the majority of the threats to the internal validity of an epidemiologic study: confounding, selection bias and information bias. Confounding, perhaps first defined by John Stuart Mill (Mill 1973 (1843)), can be understood as a "intermixture of effects," wherein the observed effect of a putative causal factor is distorted due to the effect of extraneous factors. As mentioned, in studies of neighbourhood effects on health, standard methods for confounder adjustment are debated in the literature (Diez Roux 2004). To deal with this issue, we ran four statistical models: the first two adjusting for well-defined confounders based on *a priori* graphical causal models, and the third and fourth adjusting for sets of covariates as per traditional studies on neighbourhoods and health.

By far, the biggest threat to the validity of our study involves the potential selection bias introduced into the study when recruiting participants. By sending documents inviting individuals to participate in our study, we introduced a potential for self-selection given that the reasons for one's participation may have been related to the outcome of the study (Criqui 1979; Criqui et al 1979; Rothman et al 2008). The possibility of this type of bias having been introduced into our study is exacerbated by the 14.5% estimated[1] response rate that was observed in our study. Furthermore, we could not collect relevant information on non-respondents, which prevented us from

[1] Due to communication and other problems with the data collection agency, we were limited to estimations of our response rates based on sub-samples gleaned from the entire population that we contacted for recruitment.

accurately estimating the magnitude of bias introduced (Austin et al 1981). However, we did compare the 2001 CT distributions in the proportion of: males; families with French or English as the language primarily spoken at home; immigrants; individuals with a Bachelor's degree; individuals aged 15-24; individuals aged 24-55; married / common law; single; divorced / separated / widowed; those with a household income between 0-10k, 10-20k, 20-50k, and 50-100k; as well as household size and unemployment rates to the distributions observed in our study. Using 2-sided exact binomial probability tests for proportion and a 2-sided t-test for household size, we found systematic (nondifferential) differences in the proportion of individuals aged 25-44 years, the proportion of individuals with a bachelor's degree, and the proportion of immigrants and married individuals in our sample (Table 2). Given that we restricted our analysis to individuals aged 18-55 years, the age discrepancy between our data and the census-based information is to be expected. Furthermore, it is well known that individuals with higher education tend to participate more willingly in epidemiological studies (Etter & Perneger 1997; Jooste et al 1990; Søgaard et al 2004), a feature which explains the higher level of education in our cohort.

Our sample also had a higher proportion of immigrants than the Census population. We defined "immigrants" as first-generation only by asking them about their country of birth. Those born outside of Canada were classified as "immigrants," while those born in Canada were not. It is known that first-generation immigrants are more likely to be leaner than their second generation or non-immigrant counterparts (McDonald & Kennedy 2005). Thus, if immigrant status played an important biasing role in our study, the effect would most likely have been towards the null.

The same argument can be applied to the fact that our sample had more married/common law individuals than the Census population. Married individuals have better cardiovascular health and behavioural profiles than non-married individuals (Cubbin et al 2006), thus any important biasing effects would likely have been towards the null.

Misclassification of measurements is another important source of bias in epidemiological analyses. We attempted to minimize false-negative and false-positive exposure classifications in our main effect by using buffer-zones that took into account detailed information not only on CTs in which individuals lived, but also information on those which were in their vicinity.

Some may see the fact that we did not adjust for race or ethnicity in our reported measures of association as a limitation. We did not adjust for race or ethnicity because (i) it was not determined to be a confounder based on our DAG model, and (ii) as a non-modifiable confounder, any effect that 'race' or 'ethnicity' may have would be difficult to parse out from more modifiable phenomenon such as socioeconomic status (Kaufman, 2001). The non-importance of race in our models was confirmed with an ancillary analysis that included it as a covariate. We used member of a visible minority as a proxy for race in all four of our statistical models for both outcomes. The associations between ALU and our outcomes were not changed, and the associations between the race and both outcomes were non-significant and null.

Finally, endogeneity in neighbourhood studies, the phenomenon wherein individuals choose to live in a particular neighbourhood based on particular reasons, is an important yet highly neglected issue in studies on place effects on health (Kawachi &

Subramanian 2007). We did not formally take into account the possibility that individuals were living in a particular area due to health reasons. Furthermore, ancillary analysis demonstrated that the duration of living in a given area was highly and positively associated with BMI, but not TCR. This finding points to two scenarios: The first is that those individuals who lived in an unhealthy neighbourhood for a longer duration had a higher chance of being in an unhealthy cardiovascular state (i.e., a longer exposure time leading to more severe disease). But the fact that we observed this association in BMI model and not the TCR model does fully not support this conclusion. Rather, it points to the possibility that individuals with higher BMI tended to stay in the same area of residence for longer durations than those with lower BMIs. If this was indeed the case, endogeneity may have been an important and neglected problem in our sample, given that those individuals with higher BMIs may have selected their neighbourhoods based on their condition.

8.3 GENERALIZABILITY

An important question regarding the validity of any study is with regards to the sphere in which the study results are applicable. Threats to the generalizability of any study are reduced by ensuring that the differences between the sample and the population from which it was drawn are minimal. However, as noted by Rothman et al (2008)[p. 146-7], there is an important trade-off between drawing a sample that is representative, and being able to validly answer the research questions posed. If the nonrepresentativeness is unrelated to the effects being studied, then the generalizability of the study is not compromised.

The fact that we sampled a diverse cross-section of the city's population, differing on language and socioeconomic dimensions increases the probability that our sample parallels a more realistic representation of the population as a whole. This is supported by the results of our comparison with the 2001 Census, with systematic differences observed only in four of the twenty markers we assessed.

More general, however, is the question of the relevance of our findings with respect to other metropoles around the world. Given the important cultural, economic, political and social differences between Montreal and other metropoles such as, say, Tokyo, we cannot conclude that area-level unemployment is likely associated with cardiovascular disease markers for a number of reasons. First, taking Tokyo as an example, area-level unemployment is likely not as indicative of social deprivation as in other cities, largely because of certain socio-cultural characteristics in Japan that dampen the impact of economic deprivation on the Japanese population (Wilkinson 1996). Furthermore, cardiovascular disease may unfold differently in Japanese individuals compared to Westerners given the differing nutritional resources and social, cultural and behavioural norms common to Japan (Klatsky & Armstrong 1991).

Yet there is good reason to believe that our results are generalizable insofar as area-level unemployment reflects levels of social deprivation. Numerous studies are being published which document the relevance of social deprivation to health outcomes, and our present findings are generally coherent with this norm. Although our observed associations are weak compared to typical biomedical predispositions to disease, such as smoking, they do not differ significantly from the majority of associations documented with respect to place effects on health. We did demonstrate, though informally, that

there exists a possible dose-response relationship between exposures to ALU and increased CVD risk, and there is a growing body of literature documenting plausible rationales for how social effects "get under the skin" (Krieger & Davey-Smith 2004; Taylor et al 1997). All in all, our results support the notion that higher-order social, economic, and political features of the environment can influence the health of individuals.

9. CONCLUDING REMARKS

Epidemiology, in its contemporary form, is in a state of transition. The specter of cardiovascular disease that loomed large in the mid-20th century galvanized the medical community into many lines of research. Out of the socially charged climate of the mid- to late-1960s, there grew a minor undercurrent of literature challenging established doctrine on the purely biomedical view of cardiovascular disease. This literature set the stage for an assessment of the social determinants of CVD—determinants that revolve around issues of race and ethnicity, gender, class, social justice and equal opportunity. Social Epidemiology has become a discipline which, today, challenges the relevance of decontextualized scientific research, highlights the differences between empirical fact and social or political necessity, and questions long-held notions of biological permanence and social change.

One understudied element of the social determinants of health is area-level unemployment. Despite social epidemiology's occupation with the material and social deprivation (Krieger 2001)(p. 695), and despite the fact that area-level unemployment is considered by some to be a "direct measure of urban deprivation" (Kitchen 2001)(p. 1907), remarkably little research has been done assessing the relationships between area-level

unemployment and cardiovascular disease. In the work that has been done, there is a growing consensus that area-level social deprivation plays an important role in cardiovascular disease. Furthermore, social deprivation is thought to affect men and women differently. This work, however, has been plagued with a number of methodological limitations, namely: problems of misclassification bias due to arbitrarily defined area-level units; and inconsistencies in the interpretation of associations due to unjustified use of intermediate variables as covariates. We set out to add to the literature on place effects on health while taking into consideration these known weaknesses, and investigate whether our results support the observations that social deprivation affects men and women differently.

Our findings suggest that there is an association between area-level unemployment and the antecedents of cardiovascular disease, and confirm the notion of differential sex associations. This association is gradated, indicating a potential dose-response relationship between the extent of area-level unemployment and CVD outcomes. Furthermore, the observed associations are coherent with existing literature and can be plausibly explained based on existing notions of embodiment (Krieger & Davey-Smith 2004).

Despite these justifications pointing to the plausibility of our associations, there are critical limitations of our study which preclude definitive analysis of the causal relation between social deprivation—as measured via area-level unemployment—and our chosen antecedents of cardiovascular disease. These include the cross-sectional nature of our study design, the potential for selection bias, and the possible existence of endogeneity in our cohort.

Finally, we have discussed how social indicators such as area-level unemployment fit into the larger scope of international political and economic reality. If the associations observed in our study prove causal, policy-makers will need to consider the multiple dimensions and impacts of economic development, including those which influence the health of the population.

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APPENDIX I: TABLES

Table 1a. Sample characteristics of neighbourhood study participants (n=342).

	Men (n = 169)	Women (n= 173)
Continuous Variables	Mean (Std Dev)	Mean (Std Dev)
BMI (Kg/m^2)	25.07 (3.91)	24.60 (5.18)
Age (years)	35.77 (8.87)	33.89 (8.51)
Energy expenditure (Std Total Mets)	0.12 (1.05)	-0.17 (0.857)
Fruit & Vegetable Consumption (Max = 40)	13.15 (4.86)	14.23 (4.10)
Categorical Variables	N (%)	N (%)
Unemployed		
Yes	27 (16.0)	13 (7.5)
No	142 (84.0)	160 (92.5)
Area-Level Unemployment		
Quartile 4	33 (19.5)	43 (24.9)
Quartile 3	45 (26.6)	48 (27.7)
Quartile 2	47 (27.8)	48 (27.7)
Quartile 1	44 (26.0)	34 (19.7)
Fast Food Consumption		
Yes	87 (51.5)	61 (35.3)
No	82 (48.5)	112 (64.7)
Smoker		
Never smoker/former smoker	113 (66.9)	125 (72.3)
Smoker	56 (33.1)	48 (27.7)
Education		
Less than high school	9 (5.3)	18 (10.4)
High-School completed	35 (20.7)	26 (15.0)
Trade school or university	125 (74.0)	129 (74.6)
Alcohol Consumption		
Abstainer	55 (32.5)	64 (37.0)
Moderate	80 (47.3)	97 (56.1)
Heavy	33 (19.5)	11 (6.4)
Income		
Below $20K (CAD)	44 (26.0)	57 (32.9)
Between $20K & 50K (CAD)	61 (36.1)	52 (30.1)
Above $50K (CAD)	64 (37.9)	64 (37.0)
Total Cardiovascular Risk		
0 indicators exceeding risk value	39 (22.8)	62 (35.8)
1 indicator exceeding risk value	51 (29.8)	73 (42.2)
2 indicators exceeding risk value	44 (25.7)	28 (16.2)
3 indicators exceeding risk value	28 (16.4)	9 (5.2)
4 indicators exceeding risk value	7 (4.1)	1 (0.6)

Table 1b. Number of individuals in, Mean, and Range of each ALU Quartile

ALU Quartile	N	Mean (%)	Minimum (%)	Maximum (%)
1	76	7.27	4.51	8.82
2	93	10.07	8.86	10.62
3	95	12.42	10.62	14.44
4	78	19.42	15.20	20.80

Table 1c. Cross-tabulation of ALU by BMI, TCR, and TCR sub-components for men and women

Female

	Mean Area-Level Unemployment (SD, IQR, N)											
	Q1 of ALU			Q2 of ALU			Q3 of ALU			Q4 of ALU		
	Age in Years			Age in Years			Age in Years			Age in Years		
	0-29	30-49	50+	0-29	30-49	50+	0-29	30-49	50+	0-29	30-49	50+
BMI (Kg/m2)												
0-20	7.95 (0.35, 0.49, 2)	. (., ., .)	. (., ., .)	10 (., 0, 1)	. (., ., .)	. (., ., .)	14.01 (0.54, 0.98, 3)	12.65 (1.36, 1.75, 6)	. (., ., .)	20.8 (., 0, 1)	. (., ., .)	. (., ., .)
20-25	6.88 (1.88, 3.61, 3)	7.98 (0.53, 0.83, 8)	4.53 (., 0, 1)	10.09 (0.46, 0.56, 6)	10.18 (0.47, 0.45, 22)	10.46 (., 0, 1)	11.23 (1.01, 0.81, 8)	12.94 (1.23, 1.52, 11)	. (., ., .)	19.19 (2.16, 3.05, 2)	19.74 (1.53, 0.73, 15)	20.72 (., 0, 1)
25-30	7.5 (1.66, 2.93, 3)	7.41 (0.95, 1.12, 10)	7.41 (1.87, 2.47, 4)	10.14 (0.56, 1, 3)	10.16 (0.57, 0.61, 9)	. (., ., .)	13.16 (1.43, 0.77, 5)	13.4 (1.42, 0.83, 6)	13.61 (., 0, 1)	20.8 (0, 0, 2)	19.86 (1.61, 0.85, 18)	. (., ., .)
30-35	. (., ., .)	7.7 (., 0, 1)	. (., ., .)	. (., ., .)	10.52 (0.01, 0.02, 2)	. (., ., .)	. (., ., .)	12.8 (1.9, 3.66, 3)	. (., ., .)	19.89 (., 0, 1)	18.02 (3.93, 5.55, 2)	16.81 (., 0, 1)
35+	. (., ., .)	7.57 (., 0, 1)	. (., ., .)	. (., ., .)	. (., ., .)	. (., ., .)	12.07 (., 0, 1)	13.13 (1.65, 2.33, 2)	. (., ., .)	. (., ., .)	18.61 (., 0, 1)	. (., ., .)
TCR												
0 Risk Markers	7.04 (1.53, 1.82, 4)	7.58 (0.23, 0.14, 5)	7.65 (., 0, 1)	9.67 (0.47, 0.67, 2)	10.44 (0.19, 0.19, 7)	10.46 (., 0, 1)	12.7 (1.68, 3.36, 6)	12.64 (1.58, 2.29, 5)	. (., ., .)	19.72 (1.79, 3.14, 3)	19.51 (2.33, 0.18, 5)	. (., ., .)
1 Risk Marker	7.5 (1.66, 2.93, 3)	8.18 (0.46, 0.92, 6)	5.9 (2.24, 3.96, 3)	10.03 (0.56, 1.12, 3)	9.99 (0.59, 1.18, 11)	. (., ., .)	12.03 (1.53, 2.64, 4)	13.42 (0.71, 1.24, 7)	. (., ., .)	20.8 (., 0, 1)	19.98 (1.05, 1, 11)	20.72 (., 0, 1)
2 Risk Markers	8.41 (., 0, 1)	6.66 (0.97, 1.15, 4)	. (., ., .)	10.3 (0.33, 0.57, 3)	10.17 (0.53, 0.54,	. (., ., .)	12.35 (1.94, 3.32, 4)	13.2 (1.12, 0.35,	13.61 (., 0, 1)	19.89 (., 0, 1)	19.47 (2.08, 2.18, 8)	16.81 (., 0, 1)

					10)			10)				
3 Risk Markers	. (., ., .)	7.74 (0.52, 0.74, 4)	8.79 (., 0, 1)	10.32 (0.17, 0.25, 2)	10.34 (0.31, 0.39, 4)	. (., ., .)	12.68 (0.87, 1.23, 2)	12.69 (1.84, 3.59, 5)	. (., ., .)	20.8 (., 0, 1)	19.29 (1.97, 2.19, 9)	. (., ., .)
4 Risk Markers	. (., ., .)	8.65 (., 0, 1)	. (., ., .)	. (., ., .)	10.48 (., 0, 1)	. (., ., .)	10.65 (., 0, 1)	10.67 (., 0, 1)	. (., ., .)	. (., ., .)	20.53 (0.4, 0.74, 3)	. (., ., .)
Triglycerides												
No Clinical Risk	7.38 (1.42, 1.88, 8)	7.68 (0.9, 0.65, 12)	5.62 (1.76, 3.12, 3)	10.01 (0.48, 1, 7)	10.2 (0.47, 0.49, 18)	10.46 (., 0, 1)	12.45 (1.62, 3.36, 12)	12.94 (1.13, 1.55, 11)	13.61 (., 0, 1)	19.99 (1.56, 1.61, 4)	19.99 (1.33, 0.92, 17)	20.72 (., 0, 1)
At Clinical Risk	. (., ., .)	7.63 (0.62, 1.01, 8)	8.64 (0.21, 0.3, 2)	10.28 (0.31, 0.56, 3)	10.18 (0.52, 0.62, 15)	. (., ., .)	12.07 (1.42, 2.63, 5)	13 (1.44, 2.14, 17)	. (., ., .)	20.34 (0.65, 0.91, 2)	19.39 (1.94, 3.66, 19)	16.81 (., 0, 1)
High-Density Lipoproteins												
No Clinical Risk	7.51 (1.39, 0.93, 6)	7.62 (0.29, 0.29, 8)	7.36 (1.95, 2.55, 4)	9.97 (0.53, 0.68, 4)	10.21 (0.47, 0.49, 18)	10.46 (., 0, 1)	12.7 (1.68, 3.36, 6)	13.09 (1.28, 1.52, 9)	. (., ., .)	19.72 (1.79, 3.14, 3)	19.46 (1.87, 2.33, 12)	20.72 (., 0, 1)
At Clinical Risk	6.99 (2, 2.83, 2)	7.69 (1, 1.37, 12)	4.69 (., 0, 1)	10.18 (0.39, 0.52, 6)	10.18 (0.52, 0.54, 15)	. (., ., .)	12.14 (1.48, 2.68, 11)	12.92 (1.35, 2.25, 19)	13.61 (., 0, 1)	20.5 (0.53, 0.91, 3)	19.78 (1.62, 0.95, 24)	16.81 (., 0, 1)
Total Cholesterol												
No Clinical Risk	6.75 (1.48, 2.12, 5)	7.76 (0.58, 1.11, 11)	6.94 (1.99, 3.8, 3)	9.86 (0.46, 0.51, 5)	10.2 (0.51, 0.45, 20)	10.46 (., 0, 1)	12.54 (1.59, 3.34, 13)	13.12 (1.2, 1.67, 19)	. (., ., .)	19.97 (1.35, 0.91, 5)	19.42 (1.91, 0.99, 23)	16.81 (., 0, 1)
At Clinical Risk	8.44 (0.06, 0.11, 3)	7.54 (1, 0.84, 9)	6.66 (3.02, 4.27, 2)	10.33 (0.27, 0.3, 5)	10.18 (0.46, 0.51, 13)	. (., ., .)	11.69 (1.26, 2, 4)	12.66 (1.54, 2.92, 9)	13.61 (., 0, 1)	20.8 (., 0, 1)	20.11 (1.13, 0.85, 13)	20.72 (., 0, 1)

No Clinical Risk	7.02 (1.49, 2.62, 6)	7.68 (0.5, 0.97, 15)	4.53 (., 0, 1)	10.09 (0.42, 0.56, 7)	10.16 (0.54, 0.56, 15)	10.46 (., 0, 1)	12.08 (1.45, 2.57, 9)	12.96 (1.15, 1.21, 17)	. (., ., .)	19.99 (1.56, 1.61, 4)	19.96 (1.65, 0.44, 14)	20.72 (., 0, 1)
At Clinical Risk	8.45 (0.08, 0.11, 2)	7.6 (1.41, 0.95, 5)	7.41 (1.87, 2.47, 4)	10.1 (0.57, 1.12, 3)	10.22 (0.45, 0.45, 18)	. (., ., .)	12.63 (1.66, 3.28, 8)	13 (1.58, 3.62, 11)	13.61 (., 0, 1)	20.34 (0.65, 0.91, 2)	19.49 (1.72, 0.99, 22)	16.81 (., 0, 1)

Table 1c. Cross-tabulation of ALU by BMI, TCR, and TCR sub-components for men

Male

	Mean Area-Level Unemployment (SD, IQR, N)											
	Q1 of ALU			Q2 of ALU			Q3 of ALU			Q4 of ALU		
	Age in Years			Age in Years			Age in Years			Age in Years		
	0-29	30-49	50+	0-29	30-49	50+	0-29	30-49	50+	0-29	30-49	50+
BMI (Kg/m2)												
0-20	7.63 (0.35, 0.68, 3)	7.13 (1.35, 0.54, 5)	(, ,)	9.13 (0.29, 0.57, 3)	10.19 (0.54, 0.45, 7)	. (., ., .)	12.69 (1.82, 3.6, 3)	10.69 (0.05, 0.07, 2)	. (., .,)	18.98 (., 0, .)	15.92 (1.02, 1.45, 1)	(, , 2)
20-25	6.84 (1.96, 4.08, 8)	7.35 (1.19, 0.98, 20)	(, ,)	10.23 (0.33, 0.27, 12)	9.99 (0.68, 1.11, 11)	10.46 (., 0, 1)	11.49 (1.01, 1.42, 9)	12.64 (1.56, 3.55, 15)	. (., .,)	19.22 (1.93, 0.11, .)	19.99 (0.72, 0.88, 6)	(, , 5)
25-30	7.68 (., 0, 1)	6.41 (1.53, 3, 6)	(, ,)	10.38 (., 0, 1)	9.84 (0.64, 0.89, 5)	9.5 (., 0, 1)	11.5 (1.18, 1.67, 2)	11.5 (1.46, 1.48, 8)	14.4 (., 0,)	19.34 (2.67, 2.81, 1)	19.42 (2.11, 2.78, 4)	(, , 7)
30-35	. (., ., .)	. (., ., .)	(, ,)	8.89 (., 0, 1)	9.74 (0.78, 1.5, 3)	. (., ., .)	13.99 (., 0, 1)	12.81 (1.35, 1.45, 4)	. (., .,)	20.54 (0, 0, .)	18.37 (2.92, 5.6, 2)	(, , 5)
35+	. (., ., .)	. (., ., .)	(, ,)	10.44 (., 0, 1)	10.26 (0.41, 0.58, 2)	. (., ., .)	11.79 (1.61, 2.27, 2)	12.16 (., 0, 1)	. (., .,)	20.8 (., 0, .)	15.2 (., 0, 1)	(, , 1)
0 Risk Markers	6.89 (1.64,	7.6 (1.09,	(, ,)	9.94 (0.63,	9.88 (0.68,	. (., ., .)	10.72 (0.09,	12.74 (1.65,	. (., .,)	19.21 (1.99,	20.25 (0.37,	(, , 2)

	3.21, 7)	0.81, 14)		1.07, 8)	1.43, 11)		0.18, 3)	3.58, 11)		1.56, .)	0.52, 6)	
1 Risk Marker	7.33 (1.96, 2.83, 4)	7.21 (1.16, 0.73, 12)	(, ,)	10 (0.6, 0.39, 9)	9.96 (0.66, 1.11, 11)	9.98 (0.68, 0.96, 2)	11.92 (1.38, 2.34, 11)	11.62 (1.39, 2.68, 11)	. (., .,)	19.25 (2.23, 0.76, .)	18.94 (2.1, 3.33, 5)	(, , 8)
2 Risk Markers	7.77 (., 0, 1)	5.6 (1.1, 1.2, 5)	(, ,)	. (., ., .)	10.31 (0.28, 0.53, 6)	. (., ., .)	12.9 (0.68, 0.96, 2)	11.89 (1.39, 2.75, 6)	14.4 (., 0,)	20.54 (., 0, 1)	18.63 (2.75, 5.6, 1)	(, , 6)
3 Risk Markers	. (., ., .)	. (., ., .)	(, ,)	10.38 (., 0, 1)	. (., ., .)	. (., ., .)	. (., ., .)	13.54 (0.08, 0.11, 2)	. (., .,)	20.75 (0.07, 0.1, .)	17.74 (2.96, 5.09, 2)	(, , 4)
4 Risk Markers	. (., ., .)	. (., ., .)	(, ,)	. (., ., .)	. (., ., .)	. (., ., .)	12.92 (., 0, 1)	. (., ., .)	. (., .,)	. (., ., .)	. (., ., .)	(, , .)
Triglycerides												
No Clinical Risk	7.05 (1.68, 4, 11)	7.53 (0.96, 0.79, 26)	(, ,)	9.97 (0.59, 0.96, 17)	9.92 (0.63, 1.09, 23)	9.98 (0.68, 0.96, 2)	11.66 (1.32, 1.65, 14)	12.18 (1.6, 2.96, 22)	. (., .,)	19.79 (1.51, 0.83, .)	19.24 (2.21, 2.65, 12)	(, , 12)
At Clinical Risk	7.77 (., 0, 1)	5.05 (0.58, 0.36, 5)	(, ,)	10.38 (., 0, 1)	10.4 (0.25, 0.09, 5)	. (., ., .)	12.91 (0.48, 0.96, 3)	12.3 (1.4, 2.83, 8)	14.4 (., 0,)	18.02 (3.78, 5.35, 1)	18 (2.48, 5.09, 2)	(, , 8)
High-Density Lipoproteins												
No Clinical Risk	7.19 (1.56, 0.99, 9)	7.04 (1.43, 2.69, 21)	(, ,)	9.91 (0.63, 1.06, 14)	9.91 (0.65, 1.09, 18)	9.5 (., 0, 1)	11.09 (0.94, 0.14, 6)	12.57 (1.68, 3.49, 12)	. (., .,)	18.89 (2.26, 3.37, .)	19.94 (1, 0.65, 8)	(, , 6)
At Clinical Risk	6.87 (2.13, 4.2, 3)	7.31 (0.98, 0.51, 10)	(, ,)	10.3 (0.23, 0.27, 4)	10.18 (0.5, 0.52, 10)	10.46 (., 0, 1)	12.31 (1.29, 2.69, 11)	11.97 (1.41, 2.75, 18)	14.4 (., 0,)	20.4 (0.48, 1, 1)	18.23 (2.58, 5.6, 6)	(, , 14)
No Clinical Risk	6.88 (1.68, 3.21, 10)	7.3 (1.19, 1.08, 27)	(, ,)	10.01 (0.57, 1, 14)	9.98 (0.64, 0.9, 22)	10.46 (., 0, 1)	11.89 (1.32, 2.34, 15)	12.17 (1.54, 2.91, 27)	14.4 (., 0,)	19.23 (1.99, 1.56, 1)	18.56 (2.4, 5.6, 11)	(, , 15)

At Clinical Risk HbA1c	8.24 (0.66, 0.94, 2)	5.97 (1.49, 2.57, 4)	(, ,)	9.93 (0.72, 0.79, 4)	10.1 (0.52, 0.93, 6)	9.5 (., 0, 1)	11.83 (1.55, 2.19, 2)	12.59 (1.63, 2.88, 3)	. (., .,)	20.68 (0.13, 0.26, .)	19.29 (2.33, 0.84, 3)	(, , 5)
No Clinical Risk	7.25 (1.64, 1.78, 9)	7.51 (1.14, 0.84, 16)	(, ,)	9.99 (0.64, 1.04, 11)	10.02 (0.61, 0.97, 19)	10.46 (., 0, 1)	11.9 (1.34, 1.69, 13)	12.03 (1.5, 2.76, 18)	. (., .,)	19.18 (2.21, 0.76, .)	19.11 (2.45, 3.37, 9)	(, , 8)
At Clinical Risk	6.69 (1.83, 3.19, 3)	6.72 (1.35, 2.62, 15)	(, ,)	10 (0.54, 1.05, 7)	9.97 (0.64, 0.52, 9)	9.5 (., 0, 1)	11.82 (1.33, 2.3, 4)	12.49 (1.59, 2.87, 12)	14.4 (., 0,)	20.18 (0.79, 1, 1)	18.5 (2.34, 4.7, 5)	(, , 12)

Table 2. Systematic differences between our sample and the 2001 Canadian Census population using Fisher's double-sided exact probability test.

	Proportion of the population married or common law			Proportion of the population aged 25-44		
Census Tract	Census	MNLSH Sample	p-value	Census	MNLSH Sample	p-value
1	0.29	0.45	0.00	0.34	0.62	0.00
2	0.28	0.29	0.69	0.46	0.71	0.00
3	0.44	0.68	0.03	0.42	0.73	0.00
4	0.46	0.55	0.06	0.38	0.75	0.00
5	0.43	0.48	0.44	0.46	0.68	0.00
6	0.44	0.37	0.56	0.35	0.59	0.00
7	0.49	0.65	0.03	0.26	0.43	0.02

	Proportion of the population with a University degree			Proportion of the population not born in Canada		
Census Tract	Census	MNLSH Sample	p-value	Census	MNLSH Sample	p-value
1	0.08	0.32	0.00	0.12	0.19	0.03
2	0.22	0.24	0.47	0.13	0.23	0.01
3	0.41	0.77	0.00	0.44	0.36	0.53
4	0.23	0.47	0.00	0.64	0.82	0.00
5	0.45	0.76	0.00	0.12	0.29	0.00
6	0.23	0.37	0.02	0.20	0.22	0.46
7	0.57	0.76	0.01	0.29	0.32	0.59

Table 3. Association between area-level unemployment, body mass index (BMI) and total cardiovascular risk (n=342).[a]

		Model 1[b]		Model 2[c]		Model 3[d]		Model 4[e]	
		Beta (Std Err)	p-value	Beta (Std Err)	p-value	Beta (Std Err)	p-value	Beta (Std Err)	p-value
BMI	ALU4	2.66 (0.15)	<0.001	3.20 (0.31)	<0.001	2.64 (0.17)	<0.001	1.89 (0.55)	<0.001
	ALU3	1.56 (0.19)	<0.001	2.05 (0.23)	<0.001	1.49 (0.30)	<0.001	1.39 (0.49)	0.003
	ALU2	.51 (0.17)	0.003	1.49 (0.54)	0.006	1.23 (0.36)	0.001	0.95 (0.66)	0.136
		RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value	RR (95% CI)	p-value
TCR	ALU4	1.61 (1.49-1.73)	<0.001	2.22 (1.58-3.13)	<0.001	1.91 (1.40-2.62)	<0.001	1.82 (1.35-2.44)	<0.001
	ALU3	1.47 (1.34-1.60)	<0.001	1.82 (1.42-2.34)	<0.001	1.57 (1.22-2.03)	0.001	1.66 (1.33-2.07)	<0.001
	ALU2	1.15 (1.07-1.24)	<0.001	1.42 (0.99-2.02)	0.056	1.28 (0.93-1.77)	0.132	1.37 (0.97-1.94)	0.076

[a]Referent is first (lowest) quartile throughout
[b]Model 1 included age and gender
[c]Model 2 included age, gender, and area-level education
[d]Model 3 included age, gender, area-level education, and individual education, income and employment status.
[e]Model 4 included age, gender, area-level education, individual education, income and employment status, physical activity, fast-food consumption, smoking status, fruit and vegetable consumption, and alcohol consumption.

Table 4. Odds Ratios for Total Cardiometabolic Risk Score Sub-Component Analysis.[a]

		HDL	95% CI	TRG	95% CI	TC	95% CI	HbA_1c	95% CI
Model 1[b]	ALU4	2.72	2.40-3.08	2.517	2.12-2.97	1.039	0.62-1.72	1.819	1.65-2.01
	ALU3	2.09	1.31-3.32	1.964	1.67-2.3	0.765	0.40-1.46	2.067	1.88-2.27
	ALU2	0.73	0.58-0.91	0.826	0.71-0.95	1.346	0.80-2.24	1.976	1.73-2.25
Model 2[c]	ALU4	5.93	2.07-16.95	4.933	1.64-14.81	1.465	0.68-3.12	6.32	3.61-11.04
	ALU3	4.14	1.30-13.15	1.974	1.04-3.72	0.997	0.58-1.7	2.637	1.78-3.89
	ALU2	0.93	0.76-1.12	0.979	0.71-1.34	1.592	1.01-2.53	2.739	2.33-3.21
Model 3[d]	ALU4	4.85	1.77-13.24	4.329	1.38-13.50	0.948	0.44-2	6.129	2.53-14.79
	ALU3	3.83	1.33-10.96	1.925	1.12-3.30	0.791	0.49-1.27	2.617	1.54-4.42
	ALU2	0.95	0.83-1.07	1.045	0.75-1.44	1.45	0.98-2.13	2.637	2.12-3.26
Model 4[e]	ALU4	4.19	1.18-14.84	4.505	1.05-19.24	0.987	0.46-2.09	7.455	3.78-14.68
	ALU3	2.68	0.82-8.71	1.821	0.94-3.52	0.778	0.51-1.18	2.677	1.55-4.61
	ALU2	0.61	0.46-0.79	0.985	0.50-1.92	1.404	1.25-1.57	2.852	2.19-3.71

[a]Referent is first (lowest) quartile throughout; Measures of association are expressed as odds ratios.
[b]Model 1 included age and gender
[c]Model 2 included age, gender, and area-level education
[d]Model 3 included age, gender, area-level education, and individual education, income and employment status.
[e]Model 4 included age, gender, area-level education, individual education, income and employment status, physical activity, fast-food consumption, smoking status, fruit and vegetable consumption, and alcohol consumption.

Table 5a. Gender stratified Model 4 covariates for BMI[a]

	Female BMI			Male BMI		
	Parameter Estimate	Std Err	Sig	Parameter Estimate	Std Err	Sig
Area-Level Unemployment: Q_4	2.698	2.1128	.202	1.685	1.1017	.126
Area-Level Unemployment: Q_3	2.248	1.6877	.183	.565	.7004	.420
Area-Level Unemployment: Q_2	1.707	1.5745	.278	.184	1.2069	.879
Area-Level Unemployment: Q_1 (Referent)	.	.	.	.	.	.
Area-Level Education: Q_3	-.455	1.7911	.799	-1.221	.9820	.214
Area-Level Education: Q_2	.220	1.9845	.912	-.721	1.0025	.472
Area-Level Education: Q_1 (Referent)	.	.	.	.	.	.
Age	.105	.0298	.000	.056	.0319	.078
Smoker	-.994	.6040	.100	.823	.8530	.335
Non-Smoker (Referent)	.	.	.	.	.	.
High-School	-3.557	.8351	.000	.173	.2774	.534
No High-School (Referent)	.	.	.	.	.	.
Unemployed	2.504	1.7576	.154	.397	.8059	.622
Not Unemployed (Referent)	.	.	.	.	.	.
Income: 20-50K per annum: Yes	-1.305	.7124	.067	.427	.3233	.186
Income: 20-50K per annum: No (Referent)	.	.	.	.	.	.
Income: 50k + per annum: Yes	-1.200	.6629	.070	.971	.2429	.000
Income: 50k + per annum: No (Referent)	.	.	.	.	.	.
Fruit and Vegetable Consumption	.011	.0635	.862	.083	.0719	.246
Fast Food Consumption	.833	.3610	.021	1.445	.5220	.006
No Fast Food Consumption (Referent)	.	.	.	.	.	.
Standardized Physical Activity	-1.045	.2950	.000	-.463	.3778	.220
Heavy Alcohol Consumption	-2.234	.8308	.007	-1.839	.5218	.000
Moderate Alcohol Consumption	-.025	1.1198	.982	-.148	.0849	.081
Alcohol Abstainer (Referent)	.	.	.	.	.	.

[a]Model 4 included age, gender, area-level education, individual education, income and employment status, physical activity, fast-food consumption, smoking status, fruit and vegetable consumption, and alcohol consumption

Table 5b. Association between area-level unemployment (ALU) and BMI and total cardiovascular risk (TCR) for 169 men and 173 women.[a]

		BMI				TCR					
		Men		Women		Men			Women		
		Parameter Estimate (std err)	p-value	Parameter Estimate (std err)	p-value	RR	Lower 95 % CI	Upper 95 % CI	RR	Lower 95 % CI	Upper 95 % CI
Model 1[b]	ALU4	0.71 (0.30)	0.017	4.64 (0.34)	< 0.001	1.34	1.10	1.73	2.13	1.52	2.97
	ALU3	-0.43(0.38)	0.265	3.47 (0.33)	<0.001	1.24	0.95	1.60	1.72	1.20	2.47
	ALU2	-1.75 (0.33)	< 0.001	2.50 (0.36)	<0.001	1.16	0.90	1.50	1.18	0.79	1.74
	ALU1	.	.	.	.	.	.	.	.	.	.
Model 2[c]	ALU4	0.80 (1.06)	0.457	5.74 (1.87)	0.002	1.99	1.46	2.73	2.95	0.99	8.72
	ALU3	-0.26 (0.57)	0.654	4.36 (1.31)	0.001	1.50	1.14	1.97	2.25	0.88	5.71
	ALU2	-0.35 (0.95)	0.714	3.06 (1.08)	0.005	1.25	0.78	1.99	1.51	0.70	3.26
Model 3[d]	ALU4	0.93 (1.46)	0.525	5.16 (1.96)	0.009	1.87	1.36	2.57	2.32	0.93	5.82
	ALU3	-.22 (0.84)	0.792	3.67 (1.38)	0.008	1.37	1.03	1.82	1.67	0.68	4.10
	ALU2	-0.28 (0.99)	0.772	3.18 (1.18)	0.007	1.21	0.74	1.96	1.27	0.61	2.66
Model 4[e]	ALU4	1.20 (1.45)	0.406	2.70 (2.11)	0.202	1.61	1.19	2.18	2.51	1.12	5.60
	ALU3	0.17 (0.80)	0.830	2.25 (1.69)	0.183	1.47	1.18	1.84	1.82	0.77	4.28
	ALU2	-0.02 (1.28)	0.985	1.71 (1.57)	0.278	1.26	0.82	1.94	1.41	0.74	2.70

[a]Referent is first (lowest) quartile throughout
[b]Model 1 included age
[c]Model 2 included age, and area-level education
[d]Model 3 included age, area-level education, and individual income, education and employment status.
[e]Model 4 included age, area-level education, individual income, education and employment status, smoking status, fresh fruit and vegetable consumption, fast food consumption, physical activity and alcohol consumption

Table 6a. Unemployment in the OECD, European Union, United States, Japan, and Canada[a]

	Unemployment %	
	1974-79	1980-89
OECD	4.2	7.4
European Union	4.4	7.9
United States	6.7	7.2
Japan	1.9	2.5
Canada	7.2	9.3

[a]Source: (Glyn 1995)

Table 6b. Unemployment in G7 countries, 1964-1973 and 1983-1992: Average unemployment rate for each decade, percent.[a]

	1964-73 (A)	1983-92 (B)	Ratio (B/A)
West Germany	0.79	6.03	7.63
France	2.23	9.70	4.35
Italy	5.48	10.13	1.85
Great Britain	2.94	9.79	3.33
United States	4.46	6.69	1.50
Canada	4.23	9.64	2.28
Japan	1.22	2.71	2.22

[a]Source: Reproduced from Navarro 1998, p 622.

APPENDIX II: FIGURES

Figure 1. Conceptual Framework based on Daniel et al (2008) relating area-level characteristics to cardiometabolic disease by means of a direct path through various risk markers which predispose individuals to increases in allostatic load, and through an indirect path of psychosocial and behavioural risk modifiers / mediators and factors, respectively.

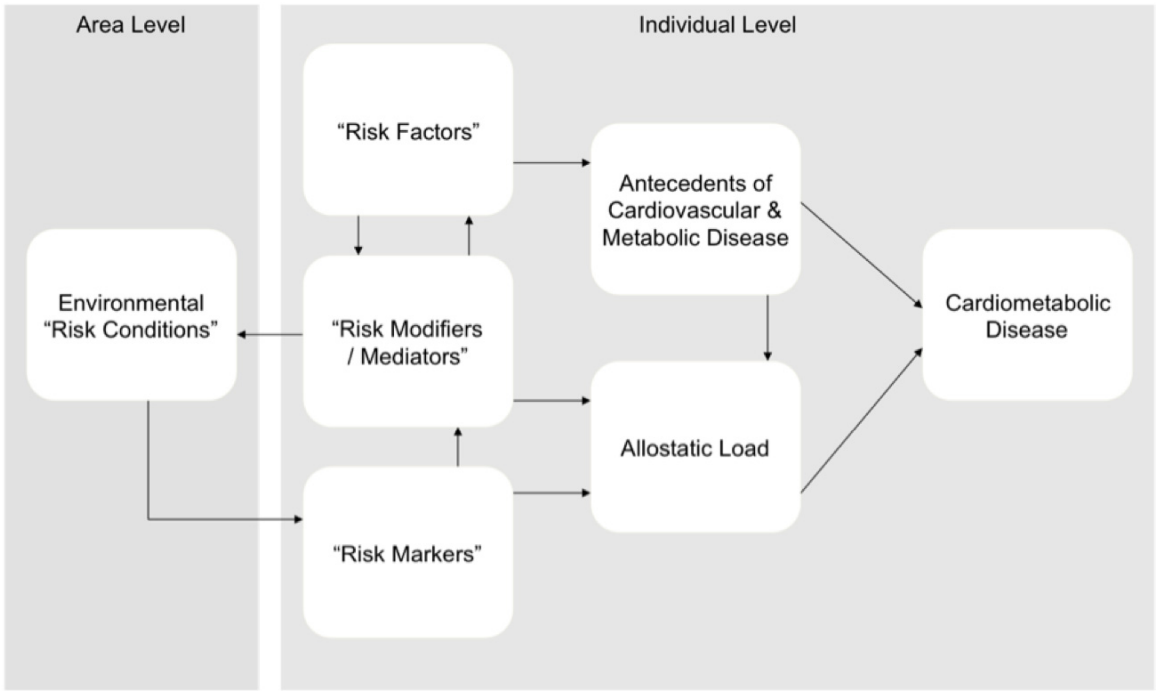


Figure 2. Theoretical causal graph based on Daniel et al. (2008) that relate structural and contextual factors (single-line boxes) to individual social (double-lined boxes), behavioural (dashed boxes) and biological (no boxes) variables. Specific pathways (not depicted) include indirect pathways that involve conscious perception of and reaction to the environment, and a direct pathway that involves non-conscious perceptions and limitations to individual socioeconomic resources. Abbreviations: ALU, Area-level unemployment; SES_A, Area-level socioeconomic status; DENS FFR, Area-level Density of Fast-Food Restaurants; DENS FVS, Area-level Density of Fresh Fruit & Vegetable Stores; FFV, Fresh Fruit & Vegetable Consumption; FF, Fast Food Consumption; EtOH, Alcohol Consumption, CHO FOOD, Consumption of High-Glycemic Index Foods.

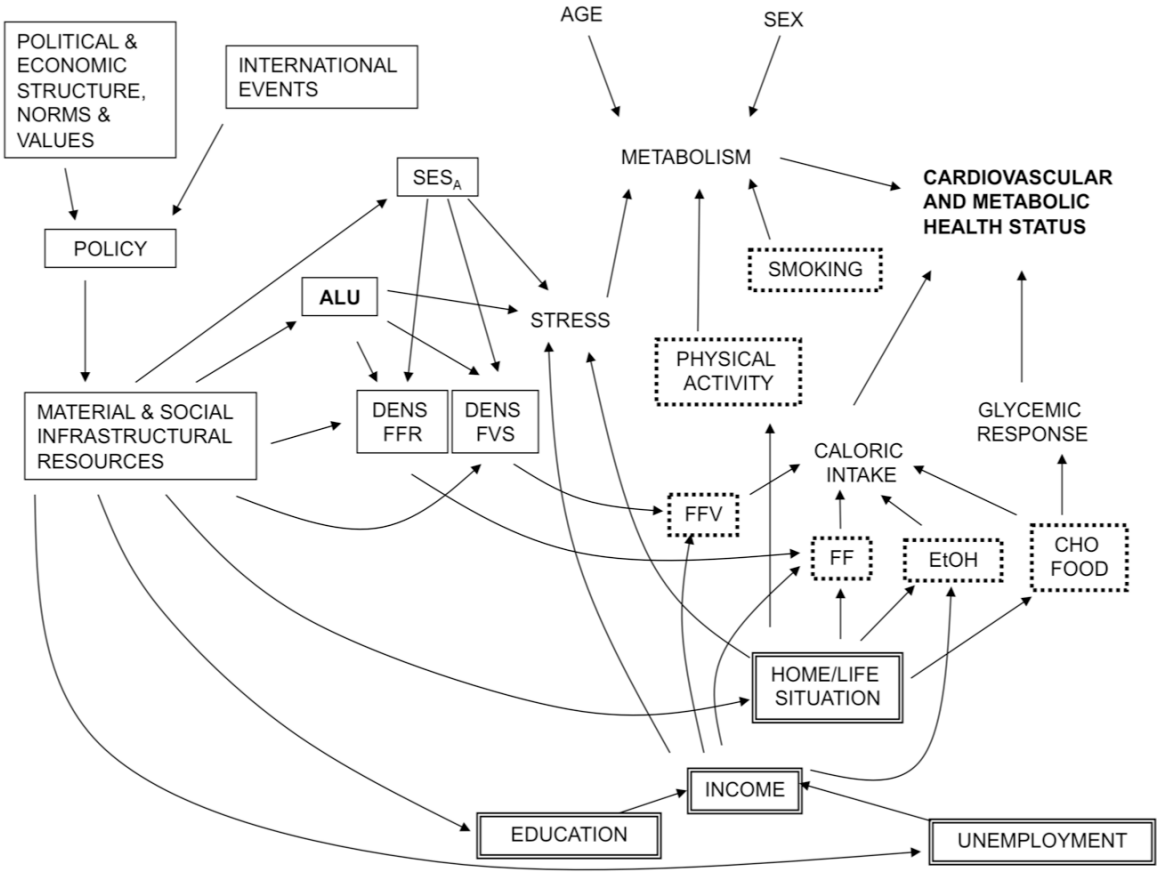


Figure 3. Analytical causal graph based on Figure 2 that relates Area-level Unemployment (ALU) to Total Cardiometabolic Risk (TCR) & Body Mass Index (BMI). Certain relations have been omitted in order to simplify the representation, including the influence of Age and Sex on socioeconomic variables such as unemployment, income and education (through discrimination), as well as on behaviours such as physical activity and alcohol consumption (through normative standards). Finally, as in Figure 2, contextual effects on individual socioeconomic factors, such as the influence of Area-level SES and Area-level unemployment on individual income, education and/or unemployment, are meant to represent structural limitations characteristic of a given area which predispose individuals to lower socioeconomic resources. Abbreviations: ALU, Area-level unemployment; SES_A, Area-level socioeconomic status; DENS FFR, Area-level Density of Fast-Food Restaurants; DENS FVS, Area-level Density of Fresh Fruit & Vegetable Stores; FFV, Fresh Fruit & Vegetable Consumption; FF, Fast Food Consumption; EtOH, Alcohol Consumption, CHO FOOD, Consumption of High-Glycemic Index Foods.

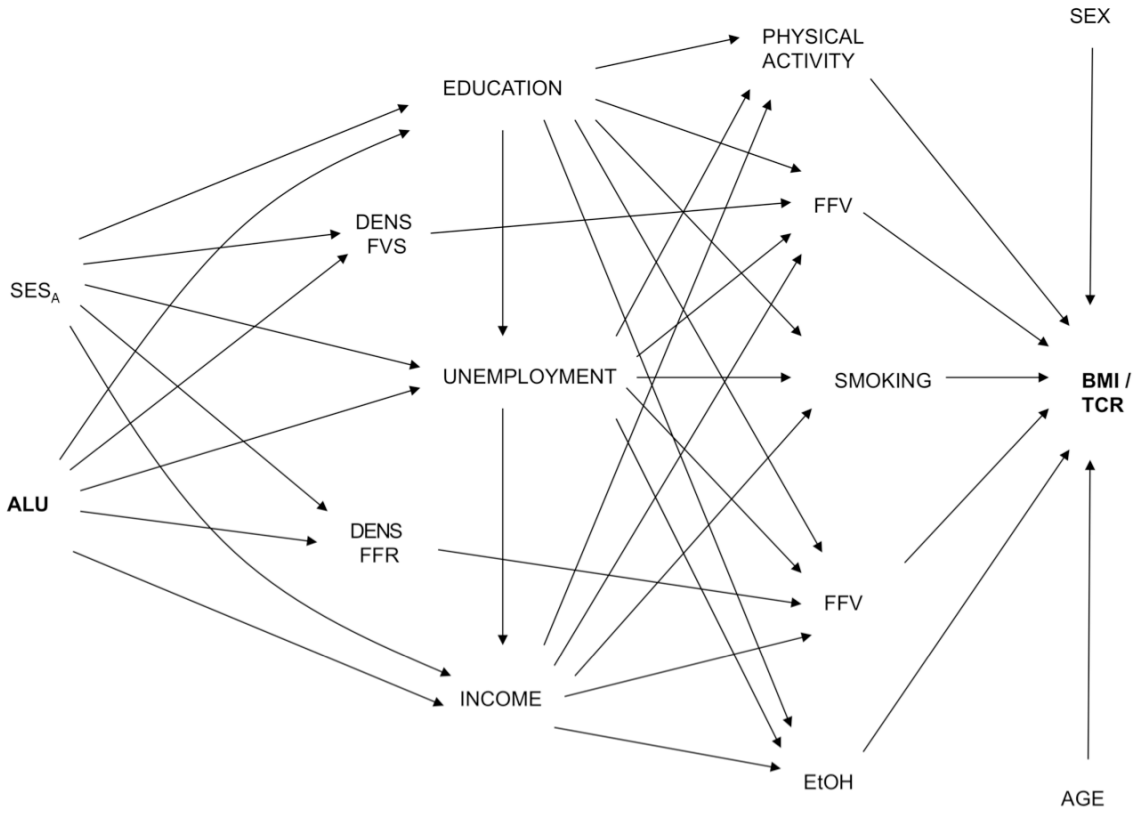


Figure 4. Hypothetical demonstration of the moving-window area technique. Individual X, while living in Census Tract (CT) number 114, is within less than 250 m of 6 other CTs, each with different area-level characteristics. Rather than taking the standard rate for CT 114 when operationalizing our area-level variables, we calculated a weighted average using MEGAPHONE© by multiplying the rate in each of the six CTs by the percent overlap within the 250 m radius (whole circle = 100 %). We then summed each weighted CT value to get a weighted average of the area-level variable of interest that was based on a 250 m radius centered on the individual. Image Source: Atlas Santé de Montréal; http://www.cmis.mtl.rtss.qc.ca/fr/atlas/caracteristiques_pop/details_carac_pop_06.html. Accessed July 8th 2008. Image is to scale.

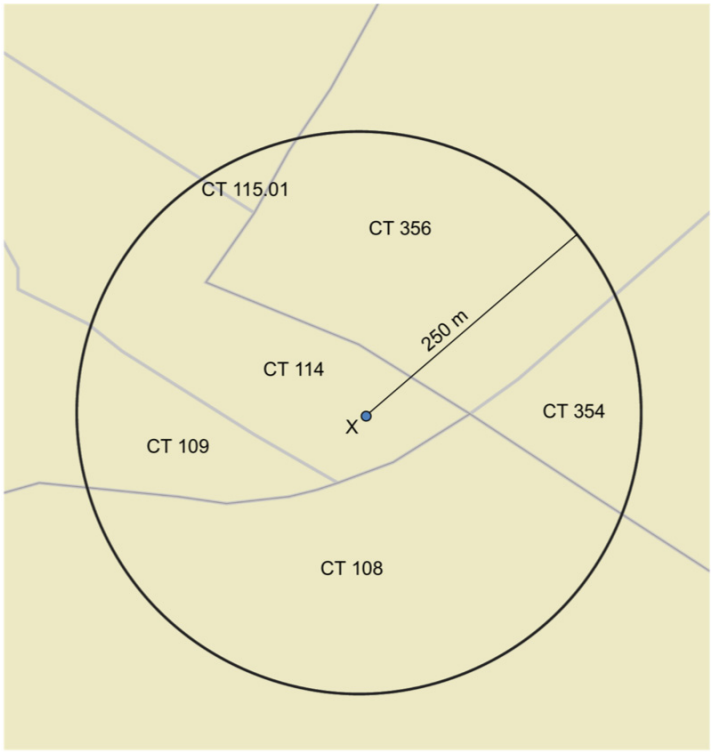
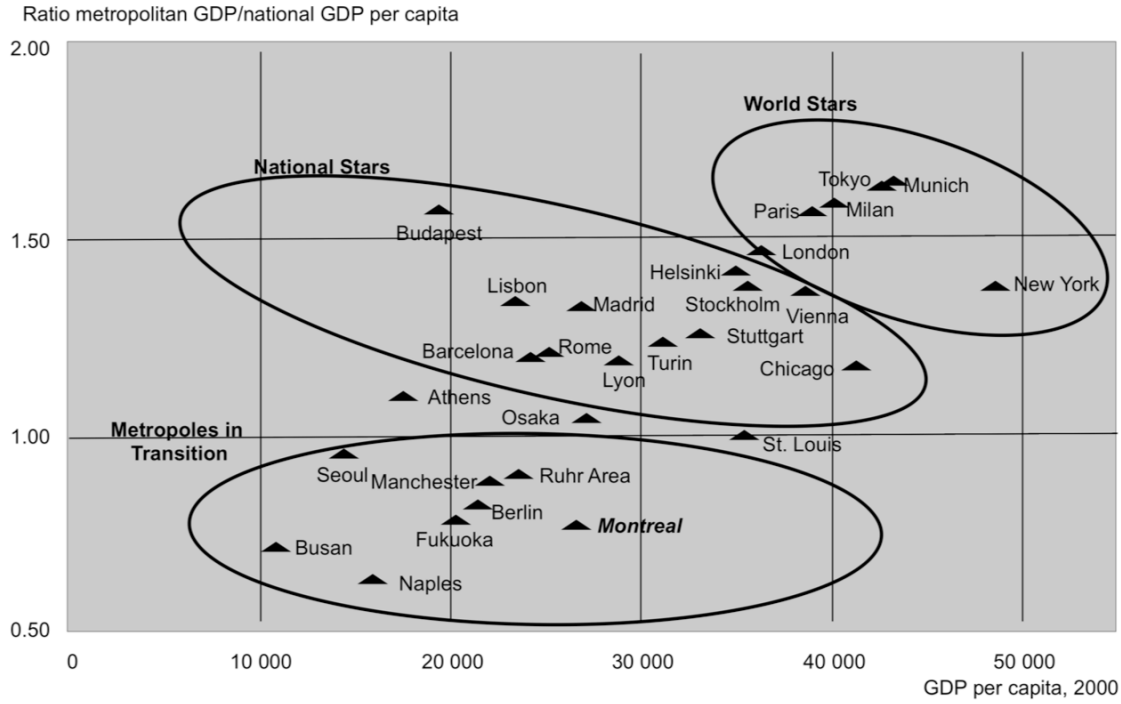


Figure 5. Absolute and relative GDPpc at Purchasing Power Parity, 2000. The figure represents the position which Montreal holds on the international scene with respect to a crude indicator of economic development. Using these standards, Montreal places 44th out of a total of 65 cities in OECD nations, and is considered a "metropolis in transition." Source: (OECD 2006)(p. 309).



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