The contributions of compositional and contextual features of local residential areas to the evolution of cardiometabolic risk over ten years in a population-based biomedical cohort

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Thesis Summary

Environmental risk conditions, compositional (collective attributes of people) or contextual (non-human attributes of areas), are associated with cardiometabolic risk (CMR). Compositional local descriptive norms (LDNs; i.e., local prevalence of a behaviour or trait) rarely feature in place and health research though they may shape attitudes that *predispose* behaviours. Simultaneously, contextual factors may *enable* predisposed behaviour. The independent and interactive effects of these different sets of influences have never been assessed.

This longitudinal research assessed the independent and interactive contributions of local descriptive norms and contextual built environment (BE) features on 10-year CMR change (ΔCMR). Three waves of South Australian adult cohort (n=4056) data were spatially linked to other datasets. LDNs were aggregated from state-wide surveillance data and expressed as buffer-specific prevalence rates for overweight/obesity, physical inactivity, and insufficient fruit intake. BE measures included walkability, public open space (POS) availability, and fast and healthful food availability.

The thesis objectives were to: 1) validate a continuous clinical index of CMR (cCICR) constructed from five risk factors, and assess its utility in estimating ΔCMR; 2) evaluate the contributions of compositional and contextual features to 10-year ΔCMR; and 3) evaluate the joint effects of compositional and contextual features to ΔCMR.

The first objective arose from the need to model a wholly clinical expression of risk that did not include non-clinical factors such as behaviour and area-level socioeconomic status. Such factors were of interest as predictors and could not be both predictors and part of the outcome. Though the cCICR exhibited moderate accuracy (AUCs 0.72 – 0.76) in predicting 10-year incident cardiometabolic disease, differential changes over time for cCICR components led to the use of a single CMR factor, glycosylated haemoglobin (HbA_{1c}) in subsequent analyses.

Analyses for Objectives 2 and 3 matched compositional norms with contextual features of the physical activity and food environments, and used multilevel models accounting for local-area

education and individual covariates (age, sex, employment status, education, marital status, and smoking status). Interactions between LDNs and BE measures were assessed.

HbA_{1c} increased over time. Local descriptive overweight/obesity, physical inactivity, and insufficient fruit intake norms predicted greater rates of HbA_{1c} increase. Greater walkability predicted a reduced rate of HbA_{1c} increase. Greater POS availability unexpectedly predicted greater rates of HbA_{1c} increase. Neither fast food nor healthful food availability were associated with change in HbA_{1c}. Local descriptive overweight/obesity norms interacted with walkability and healthful food availability. The magnitude of associations between local overweight/obesity norms and increasing HbA_{1c} was attenuated for contexts of greater walkability or healthful food availability and exacerbated for contexts of lesser walkability or healthful food availability.

This research highlights the importance of LDNs, and the multiplicative influence of context and composition that together shape CMR. Populations residing in areas with unhealthful LDNs may be resistant to many health promotion strategies, even ecological-contextual approaches such as improving walkability, given the presence of norms that do not predispose residents towards walking. Population health interventions should consider social denormalisation strategies in conjunction with policies aiming to reshape the environment to support more healthful behaviour.

Table of Contents

Thesis Summary i

Table of Contents iii

List of Tables xi

List of Figures xiii

Abbreviations and Glossary xiv

Declaration xvii

Acknowledgements xviii

1 Introduction 1

1.1 Research aim 7

1.2 Overview of the thesis 8

2 Cardiometabolic disease and risk 11

2.1 Cardiometabolic disease 12

2.2 Cardiometabolic risk 14

2.2.1 Clinical cardiometabolic risk factors 14

2.2.2 Non-clinical cardiometabolic risk factors 16

2.2.3 Composite indicators of cardiometabolic risk 20

2.3 Chapter conclusion 22

3 Theoretical foundations 24

3.1 Ecological systems 25

3.2 Composition and context 28

3.3 Collective lifestyles 31

3.4 Social norms 33

3.4.1 Injunctive and descriptive norms 33

3.4.2 Subjective and local descriptive norms 35

3.5 Interactive effects of environmental features 38
3.6 Chapter conclusion 40
4 Empirical research on environments 42
4.1 Area-level socioeconomic status 43
4.2 Composition beyond area-level socioeconomic status 46
4.3 Contextual features and cardiometabolic risk 48
4.3.1 The contextual physical activity environment and cardiometabolic risk 48
4.3.1.1 Walkability 51
4.3.1.2 Public open space 53
4.3.2 The contextual food environment and cardiometabolic risk 55
4.4 Interactions between environmental features in relation to cardiometabolic risk .. 58
4.5 Critical appraisal of the literature 60
4.6 Research aim and objectives, operational model, and hypotheses 64
5 Methods 68
5.1 Overall project design 69
5.2 Study region 70
5.3 Geographic Information Systems 71
5.4 Data sources 72
5.4.1 North West Adelaide Health Study 74
5.4.2 2007 South Australian Retail Database 77
5.4.3 2007 South Australian Property Cadastre 78
5.4.4 2006 Adelaide StreetPro™ Road Data 78
5.4.5 2007 Adelaide Public Open Space Dataset 79
5.4.6 South Australian Monitoring and Surveillance System 79
5.4.7 2006 Population and Housing Census 80
5.5 Participants: NWAHS cohort 81

5.6 Spatial units 84
5.6.1 Participant-centred road network buffers 84
5.6.2 State Suburbs 84
5.7 Measures 85
5.7.1 Study 1 85
5.7.1.1 Continuous clinical indices of cardiometabolic risk 85
5.7.1.2 Framingham Risk Scores 85
5.7.1.3 Metabolic syndrome 86
5.7.1.4 Incident type 2 diabetes, cardiovascular disease and cardiometabolic disease 86
5.7.2 Study 2 and 3 86
5.7.2.1 Outcome variable: HbA_{1c} 87
5.7.2.2 Environmental exposures 87
5.7.2.2.1 Local descriptive norms 87
5.7.2.2.2 Walkability 88
5.7.2.2.3 Public open space 89
5.7.2.2.4 Food resource availability 91
5.7.2.3 Covariates 91
5.7.2.3.1 Area-level covariate: socioeconomic status 91
5.7.2.3.2 Individual-level covariates 95
5.8 Analytic approaches 96
5.8.1 Study 1 96
5.8.2 Studies 2 and 3 98
5.9 Summary 100
6 Results 1 102
6.1 Study 1a: Validation of the continuous clinical index of cardiometabolic risk 103

Authors and affiliations 105
Abstract 106
Keywords 106
6.1.1 Background 107
6.1.2 Methods 109
6.1.2.1 Participants 109
6.1.2.2 Measures 109
6.1.2.3 Analyses 110
6.1.3 Results 112
6.1.4 Discussion 117
6.1.5 Conclusions 120
Abbreviations 120
Competing interests 120
Authors' contributions 120
Acknowledgements 121
References 122
6.2 Study 1b: Assessment of the utility of the cCICR in estimating change in cardiometabolic risk over time 124
6.2.1 Background 124
6.2.2 Methods 126
6.2.3 Results 127
6.2.4 Discussion 129
6.3 Chapter conclusion 131
7 Results 2 133
Authors and affiliations 135
Abstract 136

Keywords 137
Highlights 137
7.1 Background 138
7.2 Methods 142
7.2.1 Study area 142
7.2.2 Participants 143
7.2.3 Measures 144
7.2.3.1 Cardiometabolic risk 144
7.2.3.2 Environmental measures 144
7.2.3.3 Covariates 146
7.2.4 Analyses 147
7.3 Results 149
7.4 Discussion 155
7.4.1 Strengths and limitations 159
7.5 Conclusion 161
Competing interests 161
Author contributions 161
Acknowledgements 161
References 163
8 Results 3 167
Authors and affiliations 169
Abstract 170
Keywords 171
8.1 Background 172
8.2 Methods 175
8.2.1 Study Area 175

8.2.2 Participants 176
8.2.3 Measures 177
8.2.3.1 Cardiometabolic risk (outcome measure) 177
8.2.3.2 Environmental measures 177
8.2.3.3 Covariates 179
8.2.4 Analyses 181
8.3 Results 182
8.4 Discussion 189
8.4.1 Strengths and limitations 192
8.5 Conclusion 194
Abbreviations 194
Declarations 194
Ethics approval and consent to participate 194
Consent for publication 195
Availability of data and materials 195
Competing interests 195
Funding 195
Authors' contributions 195
Acknowledgements 195
References 197
9 Discussion 202
9.1 Objective 1 203
9.2 Objectives 2 and 3 206
9.2.1 Composition beyond area-level socioeconomic status 208
9.2.2 Conceptualisation of norms 209
9.3 Implications for policy and public health practice 211

9.3.1 Policy and legislation 211
9.3.1.1 Denormalisation strategies 212
9.3.1.2 Choice architecture 213
9.3.2 Interventions using social norms information 214
9.3.3 Geographically defined at-risk populations 215
9.3.4 Diffusion of Innovations 216
9.4 Strengths and limitations 218
9.4.1 Conceptual framework, and operational model 218
9.4.2 Causal inference 218
9.4.3 Residential self-selection 219
9.4.4 Measures 220
9.4.5 Spatial units 222
9.4.6 Residential environments as influences on health 222
9.5 Implications for advancing the research field 224
9.5.1 Expression of cardiometabolic risk 224
9.5.2 Causal inference 224
9.5.3 Composition beyond area-level SES 226
9.5.4 Social norms 226
9.5.5 Contextual built environment measures 228
9.5.6 Summative, interactive, and threshold effects 230
9.5.7 Expressions of environments 231
9.6 Conclusion 232
References 234
Appendices 267
Appendix A: PAMS Ethics Approvals 268
Appendix B: Ethic Approval for use of SAMSS data 274

Appendix C: Postcodes for NWAHS baseline sampling 276

Appendix D: Codes for Retail Database extractions 277

Appendix E: Framingham Risk Score Algorithms 278

Appendix F: Land use codes for 2007 Property Cadastre extraction 281

Appendix G: Authorship statement and copyright for manuscript 1 282

Appendix H: Authorship statement and copyright for manuscript 2 284

Appendix I: Authorship statement and copyright for manuscript 3 293

List of Tables

Table 4.1 Research hypotheses 66

Table 5.1 Database sources for individual-level and area-level information used in Studies 1-3 73

Table 5.2 NWAHS sampling frame, participant numbers for each wave and reasons for sample loss 76

Table 5.3 Predictors of loss to follow-up for the complete NWAHS cohort 77

Table 5.4 Sociodemographic characteristics of geocoded NWAHS participants at baseline compared with 2001 Population Census for the Adelaide metropolitan region 81

Table 5.5 Derivation of Study 1 analytic samples for men and women 82

Table 5.6 Derivation of analytic samples used in Studies 2 and 3 based on application of inclusion criteria 82

Table 5.7 Predictors of loss to follow-up for the analytic samples used in Studies 2 and 3 83

Table 5.8 Correlations between area-level SES variables for the included analytic samples .. 94

Table 5.9 Model fit statistics for different expressions of area-level SES in multilevel models assessing 10-year change in HbA_{1c} 95

Table 5.10 Model construction and variable inclusions for Studies 2 and 3 99

Table 6.1 Baseline characteristics of the sample according to sex 112

Table 6.2 Sample loss and sample used in models predicting each outcome among men and women 113

Table 6.3 Associations and predictive accuracies for indicators of risk predicting disease incidence 114

Table 6.4 Descriptive statistics for the cCICR, its components, and HbA_{1c} for the NWAHS cohort at each Wave 127

Table 6.5 Time (in years) predicting cardiometabolic risk as cCICR, component risk factors, and HbA_{1c} 128

Table 7.1 Derivation of analytic sample based on application of inclusion criteria 148

Table 7.2 Individual characteristics (baseline) and environmental features (Wave 2) for participants included in analyses of norms for overweight/obesity and physical inactivity. 151

Table 7.3 Associations between local descriptive overweight/obesity norm, public open space availability, walkability, and 10-year change in HbA_{1c} concentration 152

Table 7.4 Associations between local descriptive physical inactivity norm, public open space availability, walkability, and 10-year change in HbA_{1c} concentration 153

Table 8.1 Loss of analytic sample due to application of inclusion criteria 180

Table 8.2 Individual characteristics and environmental features for the analytic samples .. 183

Table 8.3 Associations between local descriptive overweight/obesity norm, food resource availability, and 10-year change in HbA_{1c} 186

Table 8.4 Associations between local descriptive insufficient fruit intake norms, food resource availability, and 10-year change in HbA_{1c} 187

List of Figures

Figure 3.1 Risk conditions position the distribution of population risk factors 27

Figure 3.2 Risk conditions shape the distribution of population risk factors 27

Figure 3.3 Conceptual framework: the multilevel influences of environmental features on cardiometabolic health including recursive relations 29

Figure 3.4 Simplified version of LW Green's PRECEDE-PROCEED Model 38

Figure 4.1 Ecological model of four domains of active living 49

Figure 4.2 Conceptual model of the influence of local food environments on dietary behaviour 55

Figure 4.3 Conceptual framework with pathways of interest to this dissertation highlighted 64

Figure 4.4 Operational model including research objectives 65

Figure 5.1 Study regions: NWAHS study area, and the urban NWAHS study area used for Studies 2 and 3 70

Figure 5.2 Graphical representation of a GIS demonstrating layers of information linked through spatial reference information 71

Figure 6.1 ROC curves for models predicting CVD, type 2 diabetes and cardiometabolic disease among men and women 116

Figure 7.1 Study area – urban north-west region of Adelaide 143

Figure 7.2 Influence of walkability on trajectories of HbA_{1c} concentration for different levels of local descriptive overweight/obesity norm 154

Figure 8.1 Study area – North-western region of Adelaide (urban area) 176

Figure 8.2 Associations between local descriptive overweight/obesity norms and HbA_{1c} trajectories according to healthful food resource availability 188

Abbreviations and Glossary

ABS	Australian Bureau of Statistics
ADA	American Diabetes Association
AIHW	Australian Institute of Health and Welfare
AIC	Akaike Information Criterion
ASSIGN	ASSessing cardiovascular risk, using SIGN: a cardiovascular risk score developed in Scotland as part of the Scottish Heart Health Extended Cohort
ATPIII	Adult Treatment Panel III
AUC	Area under the curve
BE	Built environment
BIC	Bayesian Information Criterion
BMI	Body mass index
cCICR	Continuous clinical index of cardiometabolic risk
cCICR-PCA	A cCICR constructed using principal component analysis
cCICR-Z	A cCICR constructed as the mean of standardised risk scores
CDC	Centers for Disease Control and Prevention
CI	Confidence interval
CM	Cardiometabolic
cm	centimetre
CMR	Cardiometabolic risk
ΔCMR	Change in cardiometabolic risk

CVD	Cardiovascular disease
DBP	Diastolic blood pressure
DPTI	Department of Planning, Transport, and Infrastructure
F-CVD	Framingham 10-year General CVD Risk Score
F-T2DM	Framingham 8-year Diabetes Risk Score
FPR	False positive rate
GIS	Geographic Information Systems
GPS	Global positioning system
HbA_{1c}	Glycosylated haemoglobin
HDL	High density lipoprotein
HREC	Human Research Ethics Committee
IDF	International Diabetes Federation
IEC	International Expert Committee
IPEN	International Physical Activity and the Environment Network
km	kilometre
LDL	Low density lipoproteins
LDNs	Local descriptive norms
m	metre
MAP	Mean arteriole pressure
MetS	Metabolic syndrome
NWAHS	North West Adelaide Health Study

Obj	Objective
OECD	Organisation for Economic Co-operation and Development
OR	Odds ratio
PAMS	Place and Metabolic Syndrome Project
PROS	Population Research and Outcomes Studies Unit (The University of Adelaide)
QRISK	Cardiovascular disease risk score for the United Kingdom derived from the QRESEARCH database
ROC	Receiver operating characteristics
SAMSS	South Australian Monitoring and Surveillance System
SBP	Systolic blood pressure
SCORE	Systematic COronary Risk Evaluation: a European risk score to estimate 10-year risk of fatal cardiovascular disease
SES	Socioeconomic status
T2DM	Type 2 diabetes
TPR	True positive rate
UK	United Kingdom
US	United States
WHO	World Health Organization

Declaration

I declare that:

this thesis presents work carried out by myself and does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any university; to the best of my knowledge it does not contain any materials previously published or written by another person except where due reference is made in the text; and all substantive contributions by others to the work presented, including jointly authored publications, is clearly acknowledged.

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Chapter 1

Introduction

1 Introduction

Cardiovascular disease (CVD), defined as all diseases and conditions of the heart and blood vessels, remains the biggest cause of death word-wide, accounting for 30% of all deaths (over 17.3 million) per year (Mendis, Puska & Norrving 2011). This proportion holds across Organisation for Economic Cooperation and Development (OECD) countries (32% in 2013) including Australia (31% in 2011) (AIHW 2014b; OECD 2015b). In Australia, CVD was a contributing factor for an additional 25% (total 56%) of deaths (AIHW 2014b). The absence of relevant national disease registries makes it difficult to report CVD incidence and prevalence in Australia, however, estimates for 2011-12 suggest a total of 3.7 million Australian adults (18 years and over; 21%) had CVD (AIHW 2014c). It is estimated that by 2030 over 40% of the population of the United States (US) will have some form of CVD (including hypertension), with the total direct medical costs for CVD set to triple and indirect costs estimated to rise by 61% (Heidenreich et al. 2011). For the majority of OECD countries, CVD currently accounts for the largest proportion of hospital expenditure, on average 17% of total hospital expenditure (OECD 2015a). In Australia, over 1.2 million hospitalisations (12.8% of all hospitalisations) were associated with CVD during 2012-13 (AIHW 2014a).

The average mortality rate attributable to CVD in OECD countries has decreased by over 61% since 1960 due to advances in prevention and treatment (Mendis, Puska & Norrving 2011; OECD 2015a; Tunstall-Pedoe 2003; Unal, Critchley & Capewell 2004; WHO 2011a). In Australia the CVD mortality rate has similarly fallen, with a decrease of 71% for males and 67% for females between 1981 and 2011 (AIHW 2014d, 2014b). However, prospects for further reductions in CVD disease burden within OECD countries are shrinking, even reversing in some population groups, due to rising levels of risk factors including obesity and diabetes (OECD 2015a). Moreover, CVD mortality rates have doubled in low- and middle-income countries during recent decades (Mendis, Puska & Norrving 2011). CVD is one of the four non-communicable disease groups currently targeted by the World Health Organization (WHO) to reduce the burden of non-communicable disease world-wide (Mendis & Alwan 2011; WHO 2013). Cardiovascular health was established as one of Australia's National Health Priority Areas in 1996 (AIHW 2013).

Type 2 diabetes is also being targeted as a non-communicable disease priority by the WHO, and was established as a National Health Priority Area in Australia in 1997 (AIHW 2013; Mendis & Alwan 2011; WHO 2013). Diabetes is a chronic condition defined by an excess concentration of blood glucose which increases the risk of microvascular and macrovascular damage (WHO 2006). Much of the excess mortality in type 2 diabetes is due to macrovascular complications, that is, CVD (Vasudevan & Ballantyne 2005).

The prevalence of diabetes (age-standardised) globally has nearly doubled since 1980, from 4.7% to 8.5% in 2014 (WHO 2016). In 2015 it was estimated that 415 million adults (aged 20-79 years) world-wide had diabetes (8.8%) but this is predicted to rise to 642 million (10.4%) by 2040 (IDF 2015). In Australia, an estimated 917,000 adults 18 years and over (5%) had diabetes in 2011-12 (AIHW 2014c). However, it is estimated that for every four diagnosed adult cases of diabetes in Australia, there is one undiagnosed case so the actual prevalence was likely higher (AIHW 2014d). During this same time period (2011-12), there were approximately 49,800 new cases of type 2 diabetes among individuals 10 years and over (AIHW 2014d). Though diabetes prevalence is increasing in Australia and other high income countries, the rate of increase is faster in low- and middle-income countries, and it is expected that this pattern will continue (IDF 2015; WHO 2016).

The International Diabetes Federation (IDF) have estimated that 5 million adults died due to diabetes in 2015, accounting for 14.5% of global all-cause mortality (IDF 2015). In 2011, diabetes contributed to 10% of all Australian deaths and was the sixth leading cause of death (AIHW 2014d). Diabetes was reported as the underlying cause for around 4,200 deaths, and an associated cause for 10,900 deaths (AIHW 2014d). The actual contribution of diabetes to mortality-rates was likely higher as deaths from diabetes are known to be under-reported in national statistics as diabetes is often omitted from the death certificate (AIHW 2014d; IDF 2013). Long term projections show type 2 diabetes as the leading cause of disease burden in Australia by 2023 (AIHW 2010). CVD and type 2 diabetes are preventable diseases, yet combined these two conditions represent approximately 40% of the health burden in OECD countries (OECD 2015a).

Cardiometabolic risk, the risk of developing CVD or type 2 diabetes, can be assessed using a variety of risk factors. Well established *non-modifiable* risk factors include age, sex and

genetics (Camhi et al. 2010; D'Agostino et al. 2008). Other clinical factors commonly used to indicate risk include obesity (more specifically, abdominal obesity), abnormal blood glucose levels (insulin resistance, glucose intolerance, hyperinsulinaemia), hypertension, and dyslipidaemia (Després 2008; Vasudevan & Ballantyne 2005; Wilson & Meigs 2008). These risk factors tend to cluster and are used to define metabolic syndrome by the IDF (IDF 2006) and Adult Treatment Panel III (ATPIII 2002). These clinical risk factors are influenced by lifestyle factors such as individual dietary and physical activity patterns (Camhi et al. 2010; Johnson et al. 2007; Riccardi & Rivellese 2000). Individual-level lifestyle factors including diet and physical activity, have typically been the focus of health interventions.

Interventions focused on changing individual dietary and physical activity behaviours have resulted in behaviour change, though the durability of these changes is in doubt (Greaves, CJ et al. 2011). By targeting only small groups of individuals, such interventions have limited reach, focusing on those individuals directly involved. As such, individual-level intervention strategies are unlikely to impact health across populations. Conversely, environmental factors operate at the community or population level (Daniel, Lekkas & Cargo 2010). The distribution of risk within a population is a function of environmental features such as the norms, resources, opportunities and other conditions of living (Daniel, Lekkas & Cargo 2010; Rose 1985). Individual-level risk factors are themselves shaped by these environmental features, or *environmental risk conditions*, that is, properties of environments that exacerbate a vulnerability to disease for the individuals exposed to those places (Daniel et al. 2011). Under adverse environmental conditions the entire risk distribution curve may shift unfavourably. Consequently, a greater number of individuals will be in the unfavourable tail of the risk distribution curve and considered at risk (Rose 1985). Interventions directed at these at-risk individuals will not reposition the risk distribution curve. Other individuals will become at-risk until the underlying environmental risk conditions are improved and the distribution curve is shifted more favourably (Daniel, Lekkas & Cargo 2010; Rose 1985). Though a focus on changing environmental risk conditions may have only a small effect on each individual, the effect could be substantial across populations (Rose 1985). It is therefore important to identify environmental risk conditions.

Early research assessing relationships between environmental risk conditions and health outcomes dealt mainly with area-level socioeconomic status (SES). Low area-level SES is now

well-established as associated with adverse individual-level health outcomes (Black, Moon & Baird 2014; Chaix 2009; Diez Roux & Mair 2010; Leal & Chaix 2011). Yet area-level SES tends to covary with other features of environments such as fast food availability (Black, Moon & Baird 2014). Consequently, it is necessary to assess environmental features, beyond area-level SES, to understand the mechanisms through which environments influence individual health outcomes (Mujahid et al. 2007). Research on relationships between environmental risk conditions and individual health outcomes has now evolved beyond a focus on area-level SES. However, much of the research investigating cardiometabolic risk and environmental risk conditions has been cross-sectional in design, relying on pre-defined administrative spatial areas that may not best reflect an individual's environmental exposures, and with cardiometabolic risk expressed as a single cardiometabolic risk factor, most commonly obesity, as the outcome measure. There is a need for longitudinally designed research to support causal inference, with environmental exposures expressed within participant-centred buffers to more closely approximate participant environmental exposures, and for such research to focus on cardiometabolic risk beyond obesity.

Refinement of research regarding relationships between environmental risk conditions and health outcomes has led to conceptualising environmental features as compositional (collective attributes of people) or contextual (non-human attributes of areas) (Daniel et al. 2011; Macintyre, Ellaway & Cummins 2002). The relationship between composition and context is complex. Composition and context are mutually reinforcing, each shaping the other (Cummins et al. 2007; Macintyre, Ellaway & Cummins 2002). Both compositional and contextual features are associated with cardiometabolic risk (Leal & Chaix 2011).

One group of compositional characteristics of particular interest to this dissertation is local descriptive health-related norms. In psychology, descriptive norms, or what most other people do, are recognised as important predictors of individual behaviour including physical activity and diet. Descriptive norms are included in Social Learning Theory, Social Contagion Theory (or Social Network Theory), Cialdini's Focus Theory of Normative Conduct, and more recent versions of the Theory of Planned Behaviour (Ajzen 1991; Bandura 1971; Christakis & Fowler 2013; Cialdini, Reno & Kallgren 1990; Rivis & Sheeran 2003). Descriptive health-related norms vary spatially (Blok et al. 2013). However, spatially-defined descriptive norms (i.e. *local descriptive norms*) and their associations with health outcomes, have rarely been studied.

Local descriptive health-related norms may act as predisposing factors, motivating individuals to participate in health-related behaviours thus influencing clinical health outcomes. Consequently, local descriptive norms may be of considerable relevance to preventative strategies aiming to reduce rising cardiometabolic disease rates. It is therefore necessary to empirically assess the influence of local descriptive norms on individual cardiometabolic risk.

While local descriptive norms may predispose individuals towards (or against) a behaviour, contextual built environment features may enable (or inhibit) such behaviours. Hence, built environment features may interact with local descriptive norms in relation to health outcomes that are a function of behaviour (and vice versa). For example, associations between a local descriptive norm for physical activity and cardiometabolic health may be more pronounced in areas where the built environment is supportive of physical activity, such as better walkability, as opposed to less supportive environments. This aligns with the public health tenet that to maximise healthy behaviours, environments and policy must support healthful choices, and individuals should be motivated and educated to make those choices (Sallis, Owen & Fisher 2008; WHO 1986). Only a small number of studies have assessed interactions between environmental features in relation to cardiometabolic risk. No studies have reported assessing interactions between local descriptive norms and contextual built environment features in relation to cardiometabolic risk.

If norms are to be studied, and the limitations of much existing place and health research addressed, then the definition of the outcome measure bears attention. For the assessment of cardiometabolic risk, the use of composite risk measures has been recommended over single measures (Jackson et al. 2005; Perk et al. 2012). However, well-known composite cardiometabolic risk measures include both clinical measures, such as hypertension and dyslipidaemia, and non-clinical factors such as health behaviour, family history and area-level SES (D'Agostino et al. 2008; Hippisley-Cox et al. 2007; Wilson et al. 2007; Woodward, Brindle & Tunstall-Pedoe 2007). While the inclusion of non-clinical factors may improve absolute risk prediction, it can limit the use of composite risk indicators in inferential research. Such non-clinical factors may be of interest as independent predictors of cardiometabolic risk, and consequently cannot be included as part of the outcome measure. Additionally, not all of this information may be available within secondary data sources such as population health cohorts and cross-sectional surveys.

The use of metabolic syndrome to represent cardiometabolic risk in such studies would appear a solution. However, metabolic syndrome, though defined solely using clinical measures, provides no information on risk severity due to its classification of an individual as either having or not having the syndrome (ATPIII 2002; Després 2008; IDF 2011). Constructing a continuous index of risk from clinical risk factors would more closely approximate an individual's actual risk continuum, providing information on risk severity. Such a measure would need validation, and if such a measure were to be used in longitudinal research, its performance in estimating change over time assessed. This may be of particular importance as cardiometabolic risk factors may differentially respond to environmental and behavioural exposures (Paquet et al. 2014). Though the risk factors included within a composite index may change over time, the sum total of their changes, as expressed by the composite risk index, may not.

To combat the rising rate of cardiometabolic disease, it is essential to understand the independent and interactive influences of environmental features on cardiometabolic risk. Such understanding can inform targeted intervention strategies aiming to improve population-wide health outcomes. One group of compositional features that may be important to individual cardiometabolic risk is local descriptive health-related norms. They are, however, under-researched in this regard but may offer an innovative intervention target. Moreover, associations between local descriptive norms and cardiometabolic outcomes may vary according to the features of the built environment (and vice versa). No studies have reported whether the influence of local descriptive health-related norms and contextual built environment features interact in relation to cardiometabolic risk. This dissertation contributes to the field of place and health research by addressing these research gaps.

1.1 Research aim

The aim of this research was to assess the independent and interactive contributions of local descriptive norms and built environment features on 10-year change in individual-level cardiometabolic risk.

Importantly, at the outset, assessment of the influence of environmental risk conditions on change in cardiometabolic risk requires consideration of the measure by which cardiometabolic risk is expressed.

The research aim therefore included three objectives:

1) Validation of a continuous index of cardiometabolic risk constructed from five clinical risk factors, and assessment of its utility in estimating change in cardiometabolic risk over time;

2) Evaluation of the contributions of compositional and contextual features to change in cardiometabolic risk over 10 years; and

3) Evaluation of the joint effects of compositional and contextual features to change in cardiometabolic risk.

1.2 Overview of the thesis

This dissertation includes three main studies, each presented as a separate published manuscript in Chapters 6, 7 and 8. The studies are observational in design, using 10 years (3 waves) of data from a prospective biomedical cohort, the North West Adelaide Health Study (NWAHS), comprised of over 4000 adult participants. Study 1 attends to Objective 1. Studies 2 and 3 attend to Objectives 2 and 3. For Studies 2 and 3, the NWAHS cohort data was spatially linked with other datasets using a Geographic Information System (GIS). An overview of the structure of this dissertation is provided below.

Chapter 2 outlines the pathophysiology underlying cardiometabolic disease. The chapter includes brief descriptions of individual-level cardiometabolic risk factors and composite indicators of risk.

Chapter 3 presents the theoretical reasoning underpinning the need to assess environmental risk conditions in relation to cardiometabolic risk. The complex relationship between composition and context is expanded, and the conceptual framework underlying this dissertation introduced. This chapter also defines different types of social norms and outlines the need to investigate descriptive health-related norms, expressed for local areas, in relation to cardiometabolic risk. The assessment of associations between local descriptive health-related norms and individual-level cardiometabolic risk is novel, and a key component of this dissertation. Lastly, the potential for interactive environmental exposure effects in relation to health outcomes is discussed.

Chapter 4 provides a brief review and critical appraisal of the literature regarding associations between the food and physical activity environments, and cardiometabolic risk. The chapter concludes with a restatement of the Research Aim and Objectives and presentation the research hypotheses.

Chapter 5 outlines the methods including the overall project design, defines the study regions, and describes the datasets, operationalisation of measures, and analytic strategies used to achieve the research objectives.

Chapter 6, attending Objective 1, includes into two main sections. The first section is the first published manuscript which reports the construction and validation of two continuous clinical

indices of cardiometabolic risk (cCICR). The predictive accuracies of two cCICRs are compared with that of the Framingham Risk Scores for CVD and type 2 diabetes in relation to incident cardiometabolic disease (CVD and type 2 diabetes). The second section presents the assessment of the utility of one cCICR for estimating change in cardiometabolic risk over time, comparing the cCICR with its five component risk factors and HbA_{1c}.

Chapter 7 presents the second published manuscript, addressing Objectives 2 and 3 within the physical activity environment. The study assessed the independent and interactive contributions of compositional norms (local descriptive norms for overweight/obesity and physical inactivity), and built environment features (walkability and availability of POS) on change in cardiometabolic risk as HbA_{1c}.

The third manuscript is provided as **Chapter 8**, attending Objectives 2 and 3 within the food environment with a focus on local descriptive body weight and dietary norms, and fast food and healthful food availability in relation to change in HbA_{1c}.

The final chapter, **Chapter 9**, summarises the findings as they relate to the research objectives and hypotheses, presents the implications for policy and public health practice and the strengths and limitations of the research. The chapter concludes with implications for advancing the field of research regarding environmental influences on cardiometabolic health.

Chapter 2

Cardiometabolic disease and risk

2 Cardiometabolic disease and risk

Cardiometabolic disease is a major public health issue. Individuals at-risk of developing cardiometabolic disease can be identified through assessment of clinical risk factors. Clinical risk factors can be modified by individual-level health behaviours which have typically been the focus of interventions, often ignoring the role of environmental risk conditions in shaping health behaviour. Reducing population-level cardiometabolic risk may be better achieved by environmental based interventions rather than by interventions focusing only on at-risk individuals. Environmental risk conditions, including local descriptive health-related norms, are discussed in Chapters 3 and 4. This chapter focuses on cardiometabolic disease, risk and composite risk indicators, providing background information relating to the expression of cardiometabolic risk. This directly relates to Objective 1 and is supportive of Objectives 2 and 3.

Section 2.1 briefly describes the pathophysiology underlying cardiometabolic disease. **Section 2.2** discusses individual-level risk factors for cardiometabolic disease, distinguishing between clinical and non-clinical measures, and introduces composite indicators of cardiometabolic risk. **Section 2.3** concludes the chapter.

2.1 Cardiometabolic disease

CVD is the leading cause of mortality in Australia and world-wide (AIHW 2014b; Mendis, Puska & Norrving 2011). Prominent types of CVD include coronary or ischaemic heart disease (including myocardial infarction and angina), stroke (ischaemic and haemorrhagic), and heart failure/cardiomyopathy (AIHW 2014c). The major underlying cause of CVD is atherosclerosis, and abnormal build-up of fatty streak lesions and larger fibro-fatty plaques within arteries (Greaves, DR & Channon 2002; Libby 2001). Growth of atherosclerotic plaque can significantly narrow arterial lumen leading to chronic syndromes (e.g., angina or limb claudication). Plaques may also rupture leading to thrombosis and embolism with resultant acute syndromes including myocardial infarction and stroke (Greaves, DR & Channon 2002; Libby 2001). Atherosclerosis is a pathological process beginning early in life (Greaves, DR & Channon 2002).

Diabetes is a major risk factor for CVD (Vasudevan & Ballantyne 2005; WHO 2006). Long term complications of diabetes, especially when undiagnosed or poorly controlled, include disease of the large blood vessels (macrovascular disease) potentially leading to coronary heart disease, peripheral vascular disease and stroke; and disease of the small blood vessels (microvascular disease) that can cause chronic kidney disease and failure, diabetic neuropathy (nerve damage/disease), lower limb amputations and loss of vision (AIHW 2014c; IDF 2011). Both macrovascular and microvascular abnormalities can begin before type 2 diabetes is diagnosed (Turner & Holman 1995). It is therefore important to identify at-risk individuals early.

Diabetes is caused by an inability to produce insulin (a hormone produced in the pancreas to control blood glucose levels), or by the body being unable to effectively use insulin, or both (AIHW 2014c; WHO 1999). Type 1 diabetes, estimated as 7-12% of diabetes cases in high-income countries (12% in Australia) can affect people of any age but usually develops in children or young adults (AIHW 2014c; IDF 2013, 2015). The body's own immune system damages the insulin producing beta cells in the pancreas so that insulin is no longer produced (IDF 2013). This autoimmune response is thought to result from an interplay between genetic predisposition and environmental factors, however the environmental triggers responsible (viral, dietary, or chemical) remain largely unknown (Craig et al. 2011).

Type 2 diabetes, accounting for up to 91% of adult diabetes cases in high-income countries (86% of all cases in Australia), occurs mostly in people aged 50 and over, rarely in childhood, though this is changing with increasing incidence in children and young adults (AIHW 2012, 2014c; IDF 2015). Evidence suggests that non-modifiable risk factors such as age, family history and ethnic background influence development of type 2 diabetes, along with modifiable risk factors such as obesity, physical inactivity and unhealthful diet (Shaw & Chisholm 2003), behavioural factors and traits that may be shaped by environmental features (Daniel, Moore & Kestens 2008). Hence, environmental factors are implicated in the development of both type 1 and type 2 diabetes.

The prevention of CVD and type 2 diabetes has significant potential for population-wide health improvement. An improved understanding of the factors that contribute to the development of cardiometabolic disease, environmental risk conditions in particular, is essential for the development of strategies to combat the rising prevalence of cardiometabolic disease. Assessment of cardiometabolic *risk* allows for prevention of major complications related to cardiometabolic disease and can be useful in research interested in understanding what influences the development of cardiometabolic disease.

2.2 Cardiometabolic risk

Cardiometabolic risk can be assessed using clinical measures (e.g., blood sampling; Section 2.2.1), and non-clinical measures (Section 2.2.2), including demographic (e.g., age, sex) and behavioural measures (e.g., physical activity, smoking and dietary choices). As the focus of this dissertation is not the pathophysiology of cardiometabolic disease, the measures discussed here do not form an exhaustive list. The purpose of the following sections is to provide information necessary for understanding the expression of cardiometabolic risk, particularly in relation to the literature regarding environments and cardiometabolic risk, and to highlight the need for Objective 1 of this dissertation.

2.2.1 Clinical cardiometabolic risk factors

Commonly used clinical measures of cardiometabolic risk include easy to assess risk factors such as overweight/obesity including body mass index (BMI) and central adiposity (e.g., waist circumference), and hypertension (i.e., systolic blood pressure, diastolic blood pressure and mean arteriole pressure). More invasive tests include finger prick or venous blood sampling methods to assess blood lipid concentrations (including total cholesterol, triglycerides, high density lipoproteins [HDL] and low density lipoproteins [LDL]), and measures relating to blood glucose and insulin. Cut-points have been established for these measures to identify at-risk individuals (e.g., those with hypercholesterolaemia, hypertriglyceridaemia, dyslipidaemia, hyperglycaemia, insulinaemia, and/or insulin resistance). Indeed, a clustering of these interrelated risk factors is commonly used to determine the metabolic syndrome, whose underlying pathophysiology is considered to be related to insulin resistance (ATPIII 2002; Kahn et al. 2005).

The metabolic syndrome is typically defined as hyperglycaemia, abdominal obesity, reduced HDL levels, elevated triglycerides and elevated blood pressure (ATPIII 2002). These components are associated with endothelial dysfunction, atherosclerosis, and increased risk for type 2 diabetes, vascular morbidity, and mortality (ATPIII 2002; Lakka et al. 2002). CVD and type 2 diabetes are more prevalent amongst those with the metabolic syndrome, and those individuals with the metabolic syndrome are at greater risk of developing CVD and type 2 diabetes (Isomaa et al. 2001; Lakka et al. 2002; McNeill et al. 2005; Wannamethee et al. 2005).

Type 2 diabetes, and the risk of developing type 2 diabetes, can be assessed using screening tests including fasting plasma glucose concentration, oral glucose tolerance test, and glycated haemoglobin (HbA_{1c}) concentration (Bennett, Guo & Dharmage 2007; IEC 2009; WHO 2011b). HbA_{1c} reflects the average plasma glucose level over a two to three month time period (Bennett, Guo & Dharmage 2007; Nathan, Turgeon & Regan 2007). Sampling of HbA_{1c} can be done at any time of day and does not require any special preparation such as fasting (Bennett, Guo & Dharmage 2007). This convenience has resulted in HbA_{1c} becoming the preferred test for assessing glycaemic control amongst diabetics (WHO 2011b). HbA_{1c} also has greater preanalytic stability and is less affected by day-to-day perturbations due to stress and illness than fasting plasma glucose and the oral glucose tolerance test (ADA 2015).

Though HbA_{1c} concentrations of 6.5% or above are considered indicative of type 2 diabetes (IEC 2009), prospective studies have reported strong, continuous associations between HbA_{1c} and incident diabetes (ADA 2015). The American Diabetes Association (ADA) therefore suggests an HbA_{1c} concentration of 5.7-6.5% as indicative of high risk (prediabetes) (ADA 2015). However, the International Expert Committee (IEC) recommends 6.0% be used to indicate the lower threshold of risk (IEC 2009).

HbA_{1c} is associated with microvascular and macrovascular complications arising from elevated blood glucose levels, including risk of retinopathy, microalbuminuria, nephropathy, and neuropathy (Bennett, Guo & Dharmage 2007; Nathan et al. 2013). HbA_{1c} concentration is also monotonically associated with all-cause mortality and CVD including coronary heart disease and stroke, independently of other risk factors (Eeg-Olofsson et al. 2010; Khaw et al. 2004; Nathan et al. 2013; Selvin et al. 2010). Though a threshold for cut-off values has been established for HbA_{1c} in determining type 2 diabetes, the association with CVD may be linear, and begin well below the cut point for type 2 diabetes (Eeg-Olofsson et al. 2010; Khaw et al. 2004; Nathan et al. 2013). One study however, reported a J-shaped risk curve with increased risk between HbA_{1c} concentrations of <5.0% and mortality and CVD outcomes (Selvin et al. 2010).

The clinical measures briefly outlined here provide an indication of an individual's current pathophysiological state and risk. These measures are commonly used in both clinical settings and in research assessing associations between environmental features and individual

cardiometabolic outcomes. Many of these clinical factors are themselves influenced by other factors such as the health-related behaviours of the individual.

2.2.2 Non-clinical cardiometabolic risk factors

A number of non-clinical cardiometabolic risk factors have been implicated in the development of cardiometabolic disease. Cardiometabolic risk is associated with individual, *non-modifiable risk factors* such as age, sex and genetics, and *modifiable risk factors* such as the health behaviours of the individual (Camhi et al. 2010; D'Agostino et al. 2008; Kahn et al. 2005; Leiter et al. 2011). Environmental factors have also demonstrated associations with cardiometabolic risk and disease (Leal & Chaix 2011). Such factors are discussed in Chapters 3 and 4. This section focuses on individual-level factors.

There is strong evidence that individuals that are older, male, of lower SES, particular ethnicities or cultures (e.g., Indigenous peoples, African Americans, Hispanic Americans), and with a family history of cardiometabolic disease (i.e., genetically predisposed) are at increased risk of cardiometabolic disease (AIHW 2012; ATPIII 2002; CDC 2015; Leiter et al. 2011). Though these risk factors are useful for identifying at-risk individuals and populations, they either cannot be changed or are not easily modified, and thus their utility is limited in efforts to improve an individual's cardiometabolic risk.

Individual-level *modifiable* risk factors can be useful both in identifying at-risk individuals and as a focus of intervention strategies aimed at improving cardiometabolic risk. Such risk factors are typically the health behaviours of the individual including smoking, physical activity and sedentary behaviours, and dietary behaviours such as caloric intake and diet composition. These behavioural risk factors are known to influence the clinical risk factors outlined in the previous section.

Cigarette smoking is associated with increased CVD mortality and morbidity, type 2 diabetes, glucose intolerance, insulin resistance, elevated heart rate and hypertension, chronic inflammatory state, endothelial dysfunction, and lipid abnormalities including hyperlipidaemia and low HDL levels (Benowitz 2003; CDC 2010; Willi et al. 2007). Smoking is a cause of peripheral artery disease, aortic aneurysm, coronary heart disease, stroke and cerebrovascular disease (CDC 2004, 2010) and associated with the development of atherosclerosis and induction of a hypercoagulable state creating risk of acute thrombosis

(CDC 2010). Smoking works synergistically with other cardiometabolic risk factors, increasing their adverse effects (Benowitz 2003; CDC 2010).

Regular physical activity is associated with improvements in cardiometabolic risk due to a reduction in body fat, abdominal obesity, and visceral fat (Camhi et al. 2010; Carroll, S & Dudfield 2004; Janiszewski & Ross 2007; Ross, R et al. 2004), an increase in HDL levels and decrease in triglyceride levels (Carroll, S & Dudfield 2004; Durstine et al. 2001), reduction in systolic and diastolic blood pressure with a greater reduction in hypertensive versus normal individuals (Carroll, S & Dudfield 2004), improvement in insulin action among overweight/obese individuals, and improvements in glucose tolerance (Carroll, S & Dudfield 2004). Additionally, insulin resistance is reduced in response to acute exercise (Burstein et al. 1990; Perseghin et al. 1996), and fasting plasma glucose levels are reduced among individuals with type 2 diabetes in response to a single bout of exercise (Thompson et al. 2001). Current Australian guidelines recommend that adults (18–64 years) accumulate at least 150 minutes of moderate intensity physical activity per week (Australian Government Department of Health 2014).

Conversely, sedentary behaviour (including television viewing, a common form of sedentary behaviour) has demonstrated associations with CVD (Grøntved & Hu 2011; Stamatakis, Hamer & Dunstan 2011), elevated risk for type 2 diabetes (Grøntved & Hu 2011; Hu et al. 2003), impaired glucose metabolism (Bertrais et al. 2005; Healy et al. 2007), metabolic syndrome (Bertrais et al. 2005), dyslipidaemia (Bertrais et al. 2005; Jakes et al. 2003), reduced HDL (Bertrais et al. 2005; Stamatakis, Hamer & Dunstan 2011; Wijndaele et al. 2009), hypertension (Bertrais et al. 2005; Jakes et al. 2003; Wijndaele et al. 2009), greater C-reactive protein concentration (Stamatakis, Hamer & Dunstan 2011), and obesity and weight gain (Bertrais et al. 2005; Hu et al. 2003; Jakes et al. 2003). These associations hold even after adjusting for potential confounders including physical activity level.

Regarding diet, caloric restriction is associated with a reduction in body weight, waist circumference, abdominal fat and visceral fat (Goodpaster et al. 1999; Ross, R et al. 2000; Ross, R et al. 2004), reduction in blood pressure (Fagard 2005; Neter et al. 2003), and reduction in insulin resistance (Goodpaster et al. 1999; Ross, R et al. 2000). Where caloric restriction resulted in weight loss, associations were evident with reduced triglyceride levels

(Dattilo & Kris-Etherton 1992; Goodpaster et al. 1999), increased HDL levels (after an initial decrease) (Dattilo & Kris-Etherton 1992), and reduced LDL levels (Dattilo & Kris-Etherton 1992).

Regardless of macronutrient composition, caloric reduction appears to be the most important nutritional consideration in improving cardiometabolic risk (Lau et al. 2007; Leiter et al. 2011). Aside from reducing the total amount of food consumed, caloric reduction can be achieved through changes in diet composition. Sugary drinks are the greatest contributor to added-sugar intake in the diet in the US and may promote weight gain due to a lack of reduction in subsequent meals to compensate for these liquid calories (Leiter et al. 2011; Malik, Schulze & Hu 2006). These drinks provide little nutritional value while contributing to weight gain (Duffey et al. 2010; Malik et al. 2010a). Sugar-sweetened drinks increase the risk for type 2 diabetes and CVD independently of obesity (Malik et al. 2010a; Malik et al. 2010b) and are associated with high LDL, high triglycerides, and hypertension (Duffey et al. 2010).

Fast food consumption is associated with cardiometabolic risk. Consuming fast food is positively associated with body weight and weight gain (Bowman & Vinyard 2004; French, Harnack & Jeffery 2000; Pereira et al. 2005; Rosenheck 2008) and worsening insulin resistance (Pereira et al. 2005). Fast food is usually energy dense, poor in micronutrients, low in fibre, high in glycaemic load, contains excessive amounts of refined starch and added sugars, is high in total fat and saturated fat, high in trans-fatty acids, and excessive in portion size (Bowman & Vinyard 2004; Rosenheck 2008; Stender, Dyerberg & Astrup 2007). Trans-fatty acid consumption adversely affects multiple cardiometabolic risk factors including lipid profile, proinflammatory markers, insulin sensitivity, weight gain and abdominal fatness (Kavanagh, K et al. 2007; Mozaffarian, Aro & Willett 2009). Consumption of trans-fatty acids has been associated with coronary heart disease events (Mozaffarian, Aro & Willett 2009).

Consumption of fast food is positively associated with greater intakes of calories, total and saturated fat, sodium, and sugar (including sugar-sweetened beverages) (Bowman & Vinyard 2004; French, Harnack & Jeffery 2000; Paeratakul et al. 2003; Pereira et al. 2005; Rosenheck 2008). Further, fast food consumption is associated with lower intakes of fruits and vegetables (excepting fried potato), milk, legumes, some vitamins (e.g., A, C, beta-carotene), and fibre (Bowman & Vinyard 2004; French, Harnack & Jeffery 2000; Paeratakul et al. 2003).

Review articles have concluded that regular consumption of fruits and vegetables has a protective effect against cardiovascular disease including coronary heart disease and stroke (He, FJ, Nowson & MacGregor; Mente et al. 2009; Mozaffarian, Appel & Van Horn 2011; Ness & Powles 1997). Though the evidence supporting associations between fruit and vegetable intake and type 2 diabetes is less consistent, a recent review concluded combined fruit and vegetable intake was associated with reduced risk of type 2 diabetes (Cooper et al. 2012). Of particular note, the included meta-analysis found that individuals in the highest versus lowest category (9 versus ≤ 1 portion per week) of green leafy vegetable intake had a 16% reduced risk of diabetes (Cooper et al. 2012). Finally, fruit and vegetable intake and diets that emphasise fruit and vegetable consumption are associated with reduced cardiometabolic risk (based on assessment of nine cardiometabolic risk factors) (Shi et al. 2011), reduced weight gain (He, K et al. 2004), weight loss (Svendsen et al. 2007), reduced blood pressure (Jenkins et al. 2009; Svendsen et al. 2007) and risk of incident hypertension (Forman, Stampfer & Curhan 2009), improved blood lipid profile (Jenkins et al. 2009), and improved vascular function (endothelium dependent vasodilation) (McCall et al. 2009).

Though clinical measures of cardiometabolic risk may be influenced by the health behaviours of an individual, these risk factors may respond differentially to these influences. Improvement in diet quality may change some risk factors more than others. Soft drink consumption is associated with increased risk of developing insulin resistance over four years whilst blood pressure may not change (Dhingra et al. 2007). Similarly, exercise interventions of greater than eight weeks duration reduce HbA_{1c} levels without significantly reducing BMI in adults with type 2 diabetes (Boulé et al. 2001). These differential responses may be partly attributed to differences in the rate of change of response for the various risk factors. Glycaemic benefits from physical activity are evident after a single bout (Burstein et al. 1990; Perseghin et al. 1996), though the effect of physical activity on body composition will require repeated bouts over several weeks (or months) for an effect to be measurable. Consequently, when assessing influences on cardiometabolic risk, there is a need to consider which measure best represents cardiometabolic risk in the context of the study including the length of follow-up period, and the hypothesised mechanism of influence. HbA_{1c} may provide a useful outcome measure when research is interested in a global measure of cardiometabolic risk due to the strength and shape of its independent associations with both type 2 diabetes and CVD.

Another option for expressing cardiometabolic risk may be the use of a composite indicator that includes several established risk factors.

2.2.3 Composite indicators of cardiometabolic risk

Cardiometabolic risk factors tend to cluster and individuals with multiple risk factors are at greater risk (ATPIII 2002; Jackson et al. 2005; Kahn et al. 2005). Due to this synergistic effect, the use of composite measures of cardiometabolic risk are recommended over the use of single measures (Grundy et al. 1999; Jackson et al. 2005; Perk et al. 2012). Various composite risk indicators have been developed. These indicators are typically designed to assess an individual's absolute risk of developing cardiometabolic disease and include the Framingham Risk Scores for CVD and type 2 diabetes (D'Agostino et al. 2008; Wilson et al. 2007). Such risk indicators have both clinical and research utility. However, in addition to clinical measures such as blood pressure and HDL, these composite risk indicators may include non-clinical factors such as age (e.g., Framingham10 Year General CVD Risk Calculator, SCORE, QRISK), behaviour (e.g., smoking in the Framingham 10 Year General CVD Risk Calculator, SCORE, and ASSIGN), family history (e.g., ASSIGN, Framingham Diabetes Risk), or measures of area-level disadvantage (e.g., ASSIGN, QRISK) (Conroy et al. 2003; D'Agostino et al. 2008; Hippisley-Cox et al. 2007; Wilson et al. 2007; Woodward, Brindle & Tunstall-Pedoe 2007). While the inclusion of non-clinical factors may improve absolute risk prediction, the inclusion of such factors can limit the use of composite indicators in inferential research.

The inclusion of risk factors such as age, area-level SES and individual-level health behaviours as independent variables in models predicting cardiometabolic risk may allow for the explication of the independent contributions of exposures of interest such as environmental features. As such, these risk factors cannot be included in the outcome measure. A variable cannot be both predictor and part of the outcome measure. The removal of non-clinical risk factors from established composite risk indicators would likely reduce the predictive accuracy of such indicators. Consequently, re-validation of these composite risk indicators would be necessary. Composite cardiometabolic risk indicators constructed solely from clinical factors may be of use when assessing associations between cardiometabolic risk, and environmental features, sociodemographic and behavioural factors.

One composite indicator constructed solely from clinical risk factors is metabolic syndrome. However, metabolic syndrome, defined by a count of risk factors with measures exceeding established threshold values, is a classification whereby an individual either has or does not have the syndrome, thus providing no information on the severity of risk (ATPIII 2002; Després 2008; IDF 2011). Furthermore, the use of threshold values for each of the included risk factor measures ignores the likelihood that cardiometabolic risk is a progressive function of these combined risk factors. As such, a minimal change in risk factor measures can result in a change in metabolic syndrome classification status while a large change may not (Agarwal et al. 2012). A continuous index of risk would avoid this issue and more closely approximate the individual's actual continuum of risk including information on risk severity.

A composite cardiometabolic risk indicator constructed solely from clinical measures may be useful as an outcome measure in research investigating relationships between environmental features and cardiometabolic risk while accounting for sociodemographic and behavioural factors. However, such indicators will not inform on risk-factor specific relationships with environmental or behavioural influences. Components included within composite indicators may respond differentially to environmental and behavioural influences. Though the use of composite indicators has been recommended, consideration of risk factor specific relationships may be important. The first objective of this dissertation is the validation of a continuous clinical index of cardiometabolic risk constructed from clinical measures and the assessment of its utility in estimating change over time in cardiometabolic risk. Chapter 6 reports on the construction and validation of such a measure, and the assessment of its feasibility for use in longitudinal research.

2.3 Chapter conclusion

Various clinical measures provide an indication of cardiometabolic risk. These clinical measures tend to cluster and the use of composite risk scores has been recommended (Jackson et al. 2005; Perk et al. 2012). Though many composite risk indicators have been developed, these indicators typically include non-clinical measures that can inhibit their use in inferential research. There is a need for the construction and validation of a continuous index of cardiometabolic risk that is constructed solely from clinical measures. However, clinical risk factors may respond differentially to behavioural and environmental influences. As such, the feasibility of a composite risk indicator for use in longitudinal studies would need to be assessed prior to use.

This chapter has focused on individual-level risk factors for cardiometabolic disease. These individual-level risk factors, however, are themselves shaped by environmental risk conditions, or adverse environmental features that exacerbate disease vulnerability for exposed individuals (Daniel et al. 2011). How local residential environments influence individual health is complex and the focus of the next two chapters.

Chapter 3

Theoretical foundations

3 Theoretical foundations

Clinical and non-clinical factors, the latter including health behaviours, are associated with cardiometabolic risk. These individual-level risk factors are themselves influenced by environmental risk conditions. This chapter focuses on why considering environmental risk conditions is necessary when regarding individual-level health outcomes and introduces an under-researched group of environmental factors that may be important when considering health outcomes: local descriptive health-related norms.

Section 3.1 describes ecological systems, outlining the need to consider environments when regarding individual health. In **Section 3.2**, context and composition, two important aspects of place, are defined and the framework underlying this dissertation introduced. **Section 3.3** introduces the concept of collective lifestyles, briefly touching on agent-structure relationships including social norms. **Section 3.4** defines different types of social norms, arguing the need to assess local descriptive health-related norms in relation to health outcomes such as cardiometabolic risk. The potential for interactive environmental effects is described in **Section 3.5**. **Section 3.6** concludes the chapter.

3.1 Ecological systems

Ecological models (Bronfenbrenner 1977; McLeroy et al. 1988) are commonly used in efforts to understand environmental and broader influences on individual health and behaviour. These models incorporate multiple levels of influence, directing attention to both behaviour, and individual and environmental behavioural determinants (Bronfenbrenner 1977; McLeroy et al. 1988). Bronfenbrenner (1977) conceived of the ecological environment as a nested arrangement of interdependent systems that he termed micro-, meso-, exo-, and macro-systems.

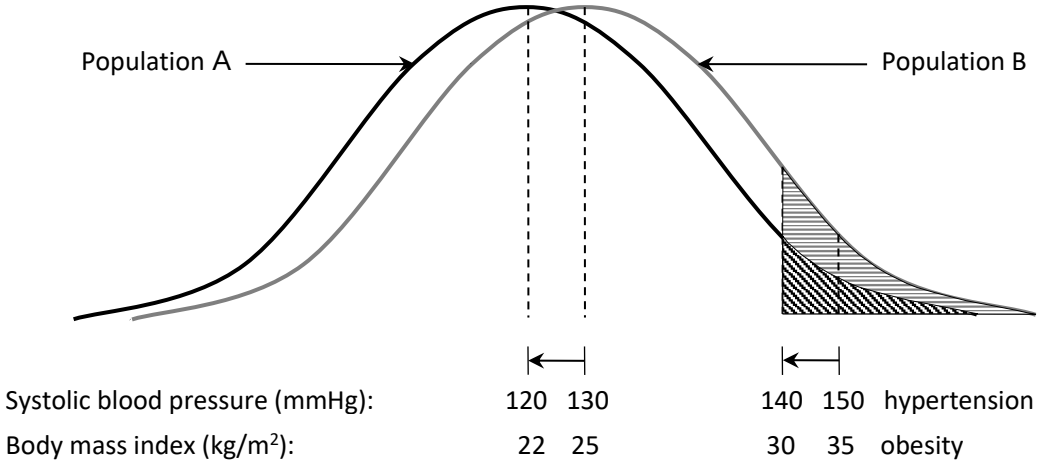
The microsystem is the complex of relationships in an immediate setting for an individual, including interactions within one's immediate family, informal networks, or work groups. The mesosystem comprises the interrelations among major settings within which the individual is involved, such as family, school or workplace, and peer groups. The mesosystem is the system of microsystems. The exosystem is an extension of the mesosystem and refers to larger social structures that do not themselves contain the individual but influence the immediate settings within which the individual is embedded. The exosystem includes the major institutions of society such as the world of work, mass media, government agencies, distribution of goods and services, communication and transportation facilities, and informal social networks. Finally, the macrosystem refers to the overarching patterns of cultural beliefs and values that influence the previously defined systems. Examples include the economic, social, educational, legal and political systems. The micro-, meso-, and exo- systems can be considered as concrete manifestations of the macrosystem (Bronfenbrenner 1977; McLeroy et al. 1988). Environmental aspects of ecological settings can be operationalised and measured to evaluate how they relate to health.

More recent conceptions of ecological systems make reference to intrapersonal (biological, psychological), interpersonal (social, cultural), organisational, community, physical environmental (e.g., topographical, climatological, built environmental), and policy levels of influence (Sallis, Owen & Fisher 2008). The inclusion of environmental and policy levels of influence distinguish these ecological models from other behavioural models and theories that emphasise the more proximal sources of influence, that is, individual characteristics and social influences of family and friends (Sallis, Owen & Fisher 2008). Ecological models have the flexibility to incorporate constructs from theories and models that focus on more proximal

levels of influence (e.g., intrapersonal and interpersonal influences), providing the opportunity to situate these within the broader influential structures provided by the more distal levels of ecological models. Moreover, ecological models suggest that complex influences on behaviour interact across these levels. For example, an individual who is highly motivated to avoid weight gain (intrapersonal level) may respond differently to walking past a strip of fast-food outlets (built attribute of the physical environment) than a less motivated individual (Sallis, Owen & Fisher 2008). It is thought that healthy behaviours are maximised when environments and policy support healthful choices, when social norms and social support for healthful choices are strong, and individuals are motivated and educated to make those choices (Sallis, Owen & Fisher 2008; WHO 1986).

Policy and environmental changes have long-term impact on the health of large numbers of people (Rose 1985). The population distribution of risk for a disease is a function of environmental risk conditions (Figure 3.1, next page) (Daniel, Lekkas & Cargo 2010; Rose 1985). In adverse environments the distribution curve may be shifted in an unfavourable direction resulting in a greater proportion of individuals (i.e., those in the tail of the curve representing risk) being considered at risk (Rose 1985). Interventions that focus on these at-risk individuals do not reposition the distribution curve, consequently other individuals will become at-risk until the underlying risk conditions (i.e., adverse environmental factors) are improved and the distribution curve is shifted in a favourable direction (Daniel, Lekkas & Cargo 2010; Rose 1985). In addition to *positioning* the population mean for individual risk factors, environments may also *shape* risk distributions and levels of risk (Figure 3.2, next page) (Daniel, Lekkas & Cargo 2010). For example, social disorder may differentially inhibit individual engagement in health behaviours between areas, resulting in greater individual variation in, for example, BMI, but the same mean value for each area (Daniel, Lekkas & Cargo 2010). It is therefore important to identify environmental risk conditions and understand how these influence health.

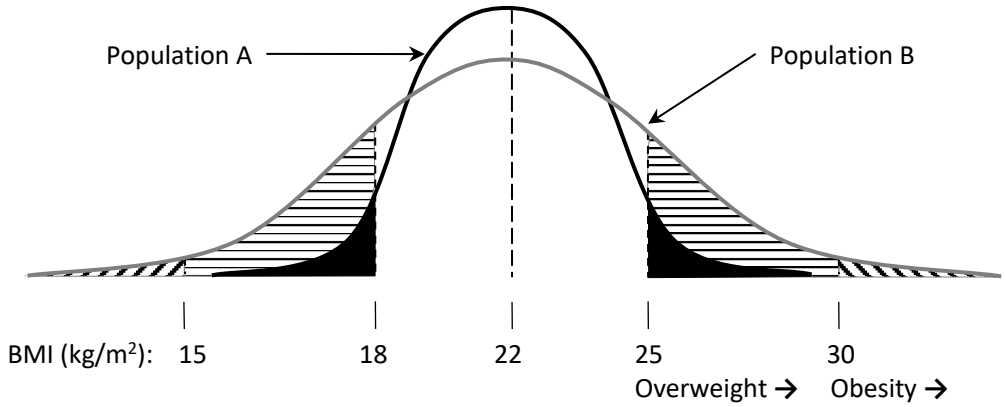
Environment: 'Risk conditions' position the distribution of risk
Individual: 'Risk factors' are expressed in the tail of the distribution



- The 'deviance' in the tails is predicted by the population mean
- 'High-risk' intervention has little effect on the population mean

Figure 3.1 Risk conditions position the distribution of population risk factors (Reproduced from Daniel, Lekkas and Cargo 2010 with permission from Elsevier)

Environment: 'Risk conditions' shape the distribution of risk
Individual: 'Risk factors' are expressed in the tails of the distribution



- Mean for Population A = mean for Population B
- Population A: 'high-risk' = overweight only, zero obesity
- Population B relative to A: 'high-risk' = 3 x prevalence of overweight + actual obesity

Figure 3.2 Risk conditions shape the distribution of population risk factors (Reproduced from Daniel, Lekkas and Cargo 2010 with permission from Elsevier)

3.2 Composition and context

Environmental risk conditions can be framed as compositional and contextual features (Daniel et al. 2011; Macintyre, Ellaway & Cummins 2002). Compositional features refer to collective attributes of a population within or between settings, and can be represented as individual-aggregated socioeconomic or sociodemographic information, or collective lifestyles and lifestyle-related norms (Daniel et al. 2011). Examples of compositional features include area-level SES, ethnicity, and local social norms including the collective health-related behaviours of residents. The compositional explanation for spatial variation in health outcomes is that people who are similar (e.g., similar SES) tend to aggregate geographically (Bernard et al. 2007). This may be intentionally, such as to share a common culture, or because a lack of personal resources (e.g., money) drives them to particular areas (Bernard et al. 2007; De Koninck & Pampalon 2007; Harvey 2010). Compositional features partly explain associations between health and place (Bernard et al. 2007).

The contextual explanation of geographic clustering in health outcomes posits that there are features of spatially defined areas that affect health beyond the contribution of compositional features (Bernard et al. 2007; Macintyre, Ellaway & Cummins 2002). These contextual features are properties of places including availability, affordability, and accessibility of health-related resources and opportunities, and physical and social conditions of living (Daniel et al. 2011; Daniel, Moore & Kestens 2008). Examples of contextual features include aspects of both the physical built environment (e.g., food availability, recreational resources, housing quality), and the social environment (e.g., crime and safety). Poorer health, including cardiometabolic risk, may develop in response to a lack of appropriate health resources such as physical activity opportunities (context), or the influence of local societal norms around physical activity behaviour (composition), or both of these environmental features simultaneously.

Difficulties in disentangling compositional and contextual effects arise as composition and context are complementary, each reflecting and reinforcing the other (Cummins et al. 2007; Macintyre, Ellaway & Cummins 2002). There is a reciprocal relationship between composition and context, each shaping the other over time (Cummins et al. 2007). The distribution of individuals across areas is neither random nor totally intentional, but reflects both opportunities and choices (Bernard et al. 2007). Where an individual lives is determined by economic means and lifestyle preferences, and may be influenced by contextual features such

as the availability of resources and services, the quality of the physical and built environments such as housing, alongside socially oriented criteria such as reputation, history or proximity to friends and family (Bernard et al. 2007; De Koninck & Pampalon 2007). Thus composition may be influenced by self-selection into areas based on context. Furthermore, context may shape composition through influences on the behaviour of residents. Contextual availability of resources may encourage or constrain individual and collective behaviour patterns. For example, greater availability of places to be recreationally physically active may encourage physical activity behaviour and manifest as a local norm for being active. Conversely, context is also shaped by composition over time as local residents influence neighbourhood resources through their consumption, service use, political processes and social connection patterns (Bernard et al. 2007; Frohlich, Corin & Potvin 2001).

Daniel and colleagues (2011) provide a framework for conceiving and framing environmental effects on health (Figure 3.3). The framework includes macro-, meso- and micro-levels of influence and illustrates both direct and indirect pathways of influence. Though published with a focus on Indigenous health, the framework is relevant to general populations.

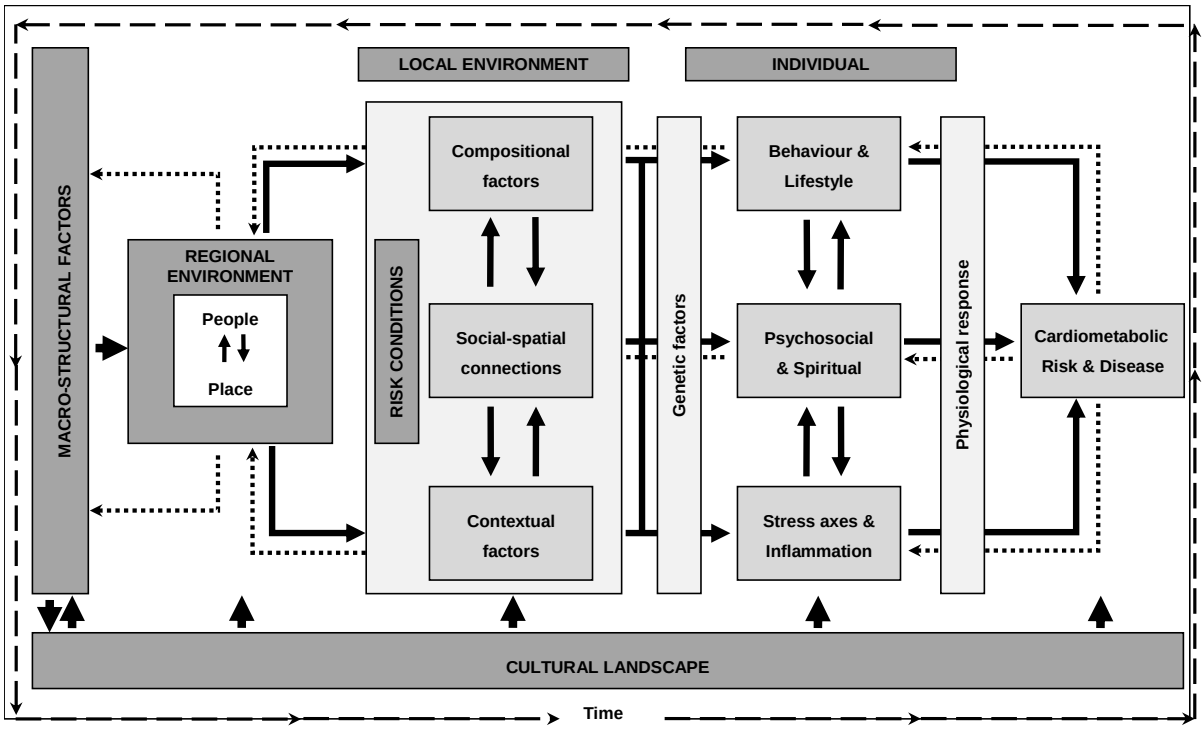


Figure 3.3 Conceptual framework: the multilevel influences of environmental features on cardiometabolic health including recursive relations
(Reproduced from Daniel et al. 2011)

Macrostructural factors shape social and physical environmental factors, creating heterogeneity of local environments. Context and composition are linked sociospatially while cultural landscapes may modify these relationships. Local compositional and contextual features influence life-course trajectories and individual-level behaviour, psychosocial factors, and stress, filtered by individual genetics.

The framework outlines pathways by which environments can influence cardiometabolic health. Compositional and contextual effects can function through both direct and indirect pathways. The deleterious effect on individual health of local air, water or soil pollutants (physical environment) represents a direct pathway. Indirect effects may function through individual-level mediators such as individual behaviour and lifestyle, or by inducing adverse psychosocial and stress responses. For example, extremes of temperature or a lack of physical activity resources may inhibit individual physical activity and thus increase cardiometabolic risk. These effects are cumulative throughout the life-course (Hertzman & Boyce 2010). Adverse conditions, such as few resources for social and emotional development, may activate genetic tendencies to cardiometabolic disease (Sunday, Eyles & Upshur 2001). Finally, the framework includes the potential for recursive effects. Environmental risk conditions in the mesosystem can be influenced by collectives of individuals, and local collectives can act on disadvantaging macrosocial factors through regional actions (Daniel et al. 2011; Reilly et al. 2008).

The differentiation of composition and context can provide a more informed understanding of how features of places and human collectives interactively affect health, supporting the design of ecological interventions (Daniel et al. 2011; Macintyre, Ellaway & Cummins 2002). For example, context may be targeted through regulatory and policy initiatives designed to increase healthful food availability while composition may be targeted by policy and organisational action to change dietary norms through nutrition education (Daniel & Green 2002; Daniel et al. 2011).

3.3 Collective lifestyles

'Lifestyle' is commonly conceptualised as a risk factor, as illustrated in the framework (Figure 3.3). The term 'lifestyle' has typically been used to represent individual-level behavioural risk factors and pathologised as a source of illness (Frohlich & Potvin 1999). 'Lifestyle' has been conceptualised as a number of discrete behaviours found to be associated with chronic or 'lifestyle' diseases (Frohlich & Potvin 1999). This 'risk factor' definition of 'lifestyle', however, may be deficient in understanding influences on individual-level health (Frohlich & Potvin 1999). This conceptualisation views lifestyle as a choice individuals make independent of their environments. It separates individuals and their behaviours from the social and situational context, stripping individual action of any contextual meaning (Frohlich 2000). Further, it conceives of lifestyle as being an individual attribute rather than one shared by collectives (Frohlich 2000).

Cockerham defines health lifestyles as "collective patterns of health-related behaviours based on choices from options available to people according to their life chances" (2005, p.55). This definition draws upon Weber's conception of lifestyle (Weber [1922] 1978) incorporating the relationship between life choices and life chances (Cockerham 2005). In this context, life choices are a proxy for agency, while life chances are a form of structure, being the opportunities people encounter in life due to their social situation (Cockerham 2005; Cockerham, Rütten & Abel 1997; Frohlich, Corin & Potvin 2001). Though lifestyle choices may be voluntary, life chances empower or constrain these choices (Cockerham 2005). People have a range of freedom but not complete freedom, their freedom is a function of the structural constraints within which they are situated (Cockerham, Rütten & Abel 1997; Frohlich 2000). Weber viewed these structural constraints as mainly economic, involving income and property, but also as rights, norms and social relationships. Chance is socially determined and social structure the arrangement of chances (Cockerham, Abel & Lüschen 1993; Frohlich 2000). Lifestyles, therefore, are not random and unrelated to structure, but are choices influenced by socially determined life chances (Cockerham, Rütten & Abel 1997; Frohlich, Corin & Potvin 2001).

Lifestyles, in Weber's conceptualisation, were not associated with individuals but with status groups. Lifestyles were considered principally a collective phenomenon (Cockerham 2005). Collective lifestyles are more than the behaviours that people engage in, representing the

relationship between people's material or sociostructural circumstances and their behaviours as choices constrained by chances (Frohlich 2000). This relationship may be shared by people in similar sociostructural circumstances, with similar influences acting on individuals experiencing similar circumstances (Frohlich 2000). Thus collective lifestyles can be considered as an expression of a shared way of relating and acting in a given environment (Frohlich, Corin & Potvin 2001). Collective lifestyles reflect and reinforce compositional and contextual conditions.

Individuals are not alone in creating and re-creating social structure through their practices (Frohlich, Corin & Potvin 2001). Collective lifestyles arise from structure-agent recursivity, producing and reproducing tastes, values and behaviours (Frohlich, Corin & Potvin 2001). Structure acts on individuals (agents) who then re-create the conditions of this structure (Frohlich, Corin & Potvin 2001). Lifestyles are structured patterns of behaviour with norms, values, and boundaries (Cockerham, Rütten & Abel 1997). Through the feedback processes of social agents, lifestyles are reproduced or transformed over time as people operationalise them (Cockerham, Rütten & Abel 1997). Norms feature within the concept of collective lifestyles, providing both the shared rules of conduct that influence individual choices, while also being recreated by the enactment of the behaviours as dictated by the norms.

3.4 Social norms

A social norm is an expectation about behaviour that is shared by a group of people (Liefbroer & Billari 2010). The definition of a social norm typically includes three components: 1) an 'ought' character, norms inform what should or should not be done; 2) the norm is shared within a group of actors, which can be of differing scales, for example, society as a whole, or subpopulations such as family, social class or religious denominations, or local communities; and 3) sanctions, which can be positive or negative, internally or externally imposed, and can vary in intensity from personal feelings of guilt or shame, gossiping and cursory remarks, through to social ostracism (Liefbroer & Billari 2010). An individual's behaviour will likely be influenced by social norms embedded within 'reference groups', the social networks or social categories they either belong to, or aspire to belong to (Liefbroer & Billari 2010).

Support for the usefulness of the concept of social norms in explaining and predicting behaviour has been mixed (Cialdini, Reno & Kallgren 1990). While some researchers assert the importance of social norms in understanding behaviour (e.g., Berkowitz 1972; Blanton & Christie 2003; Cialdini, Reno & Kallgren 1990; Fishbein 2008; Fishbein & Ajzen 1975; Liefbroer & Billari 2010; Pepitone 1976), other researchers argue that the concept of social norms is too vague, often contradictory, and ill-suited to empirical testing (e.g., Krebs 1970; Marini 1984). These criticisms have led to refinement in the definition of the concept (Cialdini, Reno & Kallgren 1990). The term, '*norm*' has had more than one meaning in both academic and everyday usage (Shaffer, 1983 in Cialdini, Reno & Kallgren 1990). The following sections provide refined definitions for social norms relevant to this thesis.

3.4.1 Injunctive and descriptive norms

Social norms are thought to characterise and guide behaviour, though not at all times or in all situations. Cialdini's Focus Theory of Normative Conduct (Cialdini, Reno & Kallgren 1990) asserts that social norms motivate behaviour when they are activated, that is, when they are salient or otherwise focused upon (Cialdini, Reno & Kallgren 1990). The theory differentiates between two types of social norms, injunctive or descriptive, either of which may be activated at a given moment. Injunctive norms refer to shared rules of conduct, what most other people approve or disapprove of. They represent what is socially sanctioned. Injunctive norms specify what *ought* to be done, enjoining action through the promise of social sanctions (Cialdini, Reno & Kallgren 1990). This is similar to the definition by Liefbroer and Billari (2010) provided

previously. Descriptive norms, however, are what most people do, what is typical or *normal* (Cialdini, Reno & Kallgren 1990). Injunctive and descriptive norms are often simultaneously available for any given behaviour (Jacobson, Mortensen & Cialdini 2011) but represent different sources of motivation (Cialdini, Reno & Kallgren 1990; Deutsch & Gerard 1955).

Injunctive norms motivate through the promise of social rewards or punishments (informal sanctions), descriptive norms inform on usual behaviour (Cialdini, Reno & Kallgren 1990; Reno, Cialdini & Kallgren 1993). Injunctive norms relate to the *inter*personal goal of building and maintaining social relationships (i.e., social approval) while descriptive norms relate to the *intra*personal goal of behaving effectively or accurately (i.e., choosing correctly) (Cialdini & Trost 1998; Jacobson, Mortensen & Cialdini 2011). However, as *what is approved of* is usually *what is done*, the meanings of injunctive and descriptive norms can easily be confused. Research indicates the benefits of distinguishing between injunctive and descriptive norms (Borsari & Carey 2001; Cialdini, Reno & Kallgren 1990; Fornara et al. 2011; Rivis & Sheeran 2003) though this distinction is often not acknowledged in literature on norms (e.g., Cheung, Chan & Wong 1999; Ewing, G 2001). There are many situations when injunctive and descriptive norms influences do not concur, such as when people approve of, but do not practice, a particular behaviour (Cialdini, Reno & Kallgren 1990). Moreover, descriptive and injunctive norms can even be antagonistic, providing conflicting information about normative behaviour in a given situation (Lapinski & Rimal 2005). For example, littering is generally socially disapproved of, yet littering appears to be quite common (Jacobson, Mortensen & Cialdini 2011). Empirical evidence supports independent and interactional effects of descriptive and injunctive norms on intentions and behaviours (Cialdini, Reno & Kallgren 1990; Kallgren, Reno & Cialdini 2000; Reno, Cialdini & Kallgren 1993; Rivis & Sheeran 2003; Smith et al. 2012).

Both injunctive and descriptive norms, though not always clearly defined, have been, and continue to be, included in predictive behavioural theories. More recent versions of the Theory of Planned Behaviour (Ajzen 1991; Rivis & Sheeran 2003) include 'subjective norms', (defined as what an individual believes others expect of them, i.e., the perceived injunctive norm in Cialdini's definition above), and descriptive norms, as predictors of behavioural intention (Rivis & Sheeran 2003). The Integrated Behaviour Model (Fishbein 2008; Montano & Kasprzyk 2008), including constructs from the Theory of Planned Behaviour and other

influential theories, has similarly incorporated descriptive norms alongside injunctive norms (Montano & Kasprzyk 2008).

Descriptive norms are also included in threshold models of collective behaviour which posit that the proportion of people engaged in a behaviour within a social system influences whether an individual will engage in that behaviour (Granovetter 1978). Lastly, Social Contagion Theory studies have found that traits (including obesity) and behaviours (including smoking) tend to cluster within social networks, and can 'spread' within these networks from person to person over time (Christakis & Fowler 2013).

3.4.2 Subjective and local descriptive norms

Descriptive and injunctive norms can be further divided according to the source of the influence, *subjective* and *local* (Carrus et al. 2009; Fornara et al. 2011). Focusing on descriptive norms, subjective descriptive norms refers to the normative influence of people who are affectively important to an individual, such as friends and family (Carrus et al. 2009; Fornara et al. 2011; Kormos, Gifford & Brown 2015). For example, Social Contagion Theory analyses behaviours and traits within social networks (Christakis & Fowler 2013). Local descriptive norms, however, are the normative influences that derive from people who are co-located (e.g., neighbours or co-workers) regardless of any emotional connections (or lack thereof) (Carrus et al. 2009; Fornara et al. 2011; Kormos, Gifford & Brown 2015). Neighbourhoods may influence behaviour directly via imitation processes, or indirectly through internalisation of norms and attitudes present within the collective lifestyles of the neighbourhood (Boardman et al. 2005).

Local descriptive norms have been associated with recycling intentions and behaviour, energy conservation behaviour, environmental conservation in hotels, environmental theft, and littering behaviour. For example, an experimental study in the US found individuals littered more in a littered environment (littering local descriptive norm) than in a clean environment (Cialdini, Reno & Kallgren 1990). Another experimental study in the US found manipulation of descriptive norm information for visitors visiting a petrified forest influenced theft of petrified wood. By providing information implying many people steal petrified wood on signs within the forest, there was an increase in petrified wood theft (Cialdini et al. 2006).

An Italian study found perceived local descriptive norms were associated with intention to recycle household waste, independently from Theory of Planned Behaviour predictors (Carrus et al. 2009). Similarly, a United Kingdom (UK) study found perceived local descriptive recycling norms were associated with recycling intentions and self-reported recycling behaviour when added to models containing Theory of Planned Behaviour constructs, strengthening model prediction (Nigbur, Lyons & Uzzell 2010). An experimental study in the US (California) found local descriptive recycling norms information provided as door-hangers, influenced the amount and frequency of recycling behaviour of individuals (Schultz 1999). A further Italian study compared the influence of four types of norms (local descriptive, subjective descriptive, local injunctive, subjective injunctive) on recycling intentions, finding descriptive norms (both subjective and local) were associated with recycling intentions more strongly than injunctive norms (both types) (Fornara et al. 2011).

In relation to energy conservation, a Californian study found descriptive normative beliefs about the energy conservation behaviours of local others (i.e., neighbours, local residents, Californians) were more strongly associated with self-reported energy conservation behaviour than were other relevant beliefs such as saving money, or protecting the environment (Nolan et al. 2008). The follow-up intervention study involving door-hangers with energy conservation messages, found local descriptive normative messages (that their neighbours conserved energy) spurred people to conserve more energy (based on meter readings) than the other messages traditionally considered to be motivational (e.g., based on self-interest, environmental protection, or social responsibility) (Nolan et al. 2008). A different Californian study (Schultz et al. 2007) on household energy consumption found the provision of local descriptive norms information along with individual household energy consumption information influenced energy consumption such that households using more than the local norm decreased their usage. However, households using less than the local norm increased their usage. The inclusion of injunctive norm information eliminated this unwanted "boomerang" effect (Schultz et al. 2007).

There are also a number of studies in hotels relating to towel usage. For example, a US experimental study found descriptive norm information on reuse of hotel room towels (i.e., majority of other hotel guest reuse their towels) increased the rate of towel reuse compared to industry standard pro-environment messages (i.e., save the environment, reuse your towel)

in hotel rooms (Goldstein, Cialdini & Griskevicius 2008). In addition, use of descriptive norm messages specific to that locale (hotel room) resulted in greater increase in towel reuse than descriptive norms messages designed to focus attention on social identity categories (men, women, citizens, hotel guest) or the standard pro-environmental plea (Goldstein, Cialdini & Griskevicius 2008).

Though local descriptive norms have been assessed in relation to environmental behaviours such as recycling and energy conservation, little attention has been given to local descriptive norms within health research. One US cross-sectional study of adults reported individuals were more likely to be obese if they reside in a community in which a relatively high proportion of the residents are obese in models including individual sociodemographic information, and area-level SES and racial composition information (Boardman et al. 2005). A more recent longitudinal study (13 years of follow up) of Dutch adults reported increased odds of an individual becoming overweight if they lived in a neighbourhood with a greater prevalence of overweight residents in models adjusted for age, sex, education and neighbourhood deprivation (Blok et al. 2013). Individuals were also more likely to quit sport if residing in an area of low sports participation, and to quit smoking in areas with low smoking prevalence. However, the sports and smoking associations did not remain after inclusion of covariates (Blok et al. 2013). The results of these studies suggest that a greater concentration of individuals within a neighbourhood who are overweight or obese may lead to an overarching acceptance of larger body size as collective attitudes dismiss (directly or indirectly) or discredit the potential harms of being overweight or obese (Boardman et al. 2005). Unfortunately, neither of these studies included in their analysis models features of the neighbourhood built environment that could potentially be associated with both the local descriptive body weight norm and individual body weight and behaviour. Built environment features such as availability of healthful and unhealthful food may be associated with both the local descriptive norm and change in individual-level health behaviours related to BMI over time. Furthermore, environmental features may interact with other environmental features in relation to individual-level health outcomes.

3.5 Interactive effects of environmental features

The PRECEDE-PROCEED Model (Green & Kreuter 2005), shown in Figure 3.4, frames antecedents of behaviour as *predisposing*, *reinforcing* and *enabling*. *Predisposing factors* are behavioural antecedents that can motivate behaviour or behaviour change. *Reinforcing factors* provide rewards or incentives to continue performing the behaviour. Finally, *enabling factors* are behavioural antecedents that facilitate and allow for the realisation of a given motivation, to allow enactment of a desired activity. Notably, more healthful environments can also be shaped by enabling factors driven by policy, organisational and regulatory actions. Predisposing, reinforcing and enabling factors collectively shape health as a function of behavioural and environmental influences. The PRECEDE-PROCEED model can be used to explain health and quality of life, and to inform intervention strategies intending to improve health and quality of life (Carlson Gielen et al. 2008; Green & Kreuter 2005).

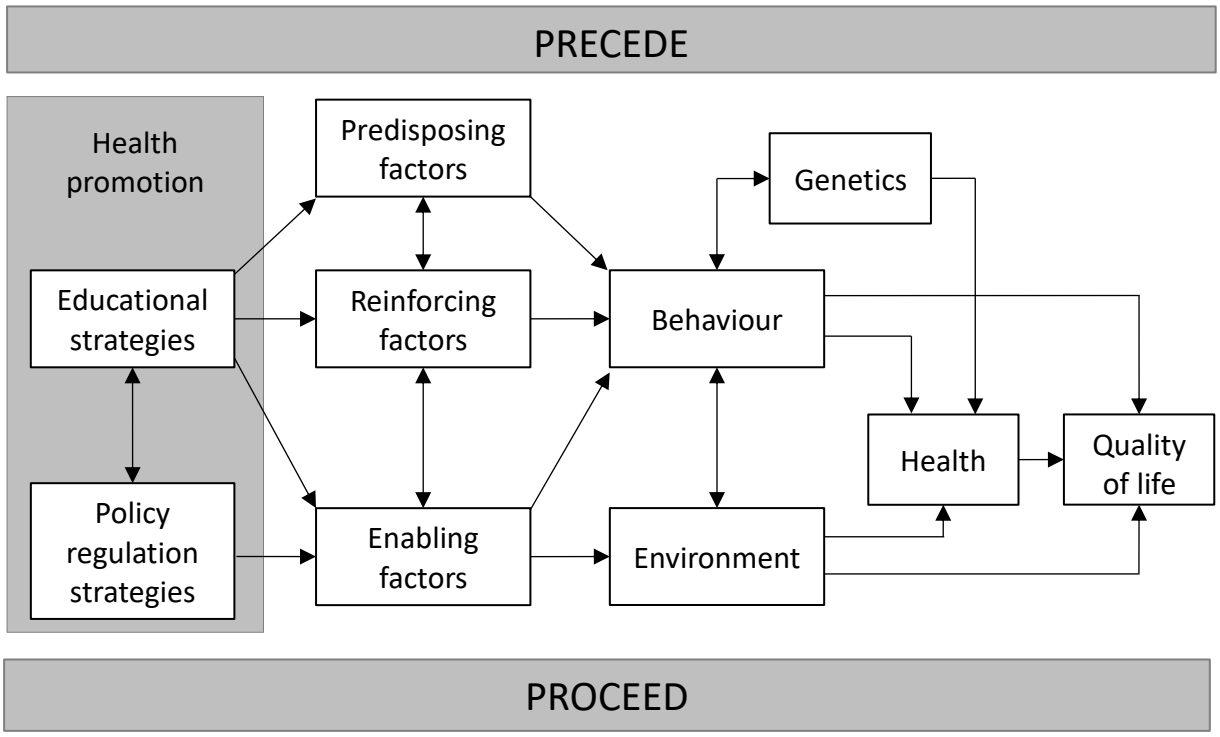


Figure 3.4 Simplified version of LW Green's PRECEDE-PROCEED Model (Adapted from Carlson Gielen et al. 2008)

Drawing on the PRECEDE-PROCEED Model, local descriptive norms can be conceptualised as enabling environmental factors that 1) directly influence behaviour as well as 2) indirectly influence behaviour via impacts on individual-level reinforcing and predisposing factors. Greater local descriptive norms for healthful behaviour may induce individuals to move

through the Transtheoretical Model's stages of change (Prochaska & Velicer 1997) from *pre-contemplation* to considering adopting the behaviour (*contemplation* and *preparation*) thus *predisposing* an individual towards the behaviour of interest. Enabling factors also drive the provision of opportunities to perform the behaviour, thus allowing an individual to reach the *action* and *maintenance* stages of change. Enabling factors of local environments include built environment features such as walkability and the availability of food and public open space (POS). Greater availability of food within a local area may enable individuals to obtain their desired foods as indirectly predisposed by local descriptive norms. Greater levels of walkability and availability of POS provide opportunities for walking and other physical activities. Consequently, it is likely that cardiometabolic risk varies according to the influences of both local descriptive norms *and* environmental factors that together shape health. Further, associations between local descriptive norms (indirect predisposing factors) and cardiometabolic risk may vary according to the level of opportunities for healthful (or unhealthful) behaviours (enabling factors). That is, features of the built environment may modify the relationship between local descriptive norms and cardiometabolic outcomes, and vice versa.

Few studies have assessed interactions between environmental features in relation to cardiometabolic outcomes, though these interactions are often represented in conceptual frameworks (e.g., Figure 3.3). The need for more studies assessing environmental interaction effects in relation to cardiometabolic outcomes (and its behavioural antecedents such as walking), has been highlighted in reviews (e.g., Leal & Chaix 2011; Lovasi, Grady & Rundle 2012; Schüle & Bolte 2015). Understanding the interactive effects of environmental risk conditions would support the design of locally-tailored interventions that exploit the combined effects of environmental features on health outcomes. No studies have assessed environmental interaction effects that include health-related local descriptive norms in relation to health outcomes.

3.6 Chapter conclusion

Environmental influences on health are important. Without consideration of environments, studies of individuals assume individuals are disconnected from one another and from their environments (Frohlich, Corin & Potvin 2001; Susser 1999). Interventions targeting environmental risk conditions can influence a greater proportion of individuals than interventions targeting at-risk individuals. As such, there is a need to understand which features of environments can act as levers in improving individual-level health. Furthermore, environmental features likely act synergistically on population health. An understanding of interactions between environmental risk conditions is therefore needed to support locally-tailored intervention design.

Social norms are important influences on individual-level behaviours, featuring in the Theory of Planned Behaviour (Ajzen 1991) and Social Contagion Theory (Christakis & Fowler 2013). However, spatial variation in social norms has rarely been investigated in relation to health outcomes. Assessment of the independent and interactive contributions of local descriptive health-related norms and contextual built environment in relation to change in cardiometabolic risk, is the aim of this dissertation. The next chapter summarises the literature regarding environmental risk conditions and cardiometabolic risk.

Chapter 4

Empirical research on environments

4 Empirical research on environments

Acting on environmental features can reshape the risk distribution for population cardiometabolic risk such that a smaller proportion of the population is at increased risk (i.e., in the tail of the distribution). It is therefore imperative that we know which environmental features relate to health outcomes. This chapter provides a brief review of the literature regarding environmental features and cardiometabolic outcomes.

Section 4.1 begins the discussion on associations between environmental features and health, with a focus on area-level SES. **Section 4.2** reiterates the definition of composition used in this dissertation and outlines the need to assess compositional features beyond area-level SES. **Section 4.3** summarises literature on the contextual physical activity and food environments, including frameworks for their conception. In **Section 4.4**, findings relating to interactions between environmental features are summarised. **Section 4.5** critically appraises the literature including discussion on issues relating to choice of spatial unit. **Section 4.6** concludes the chapter by repeating the research aims and hypotheses, presenting the operational model and defining the research hypotheses.

4.1 Area-level socioeconomic status

Since the early 1990s interest in the influence environmental risk conditions have in shaping individual-level health outcomes has grown (Bernard et al. 2007; Diez Roux 2001; Macintyre, Ellaway & Cummins 2002). Associations between environments and health outcomes have been reported for a range of health outcomes using various types of spatial units and a variety of different scales (Bernard et al. 2007; Black, Moon & Baird 2014; Chaix 2009; Diez Roux 2001; Diez Roux & Mair 2010; Leal & Chaix 2011).

Much of the early research into environmental risk conditions focused on the socioeconomic conditions of residential areas (area-level SES). Area-level SES, either as an aggregated measure of the SES of residents, such as area-level income, education, unemployment, or expressed as a composite index of area deprivation, is consistently related to mortality rates, cardiometabolic disease and risk, and other health outcomes and behaviours (Black, Moon & Baird 2014; Chaix 2009; Diez Roux & Mair 2010; Leal & Chaix 2011).

The use of aggregate measures of SES has, however, sometimes resulted in ambiguity regarding the conceptualisation of area-level SES. Aggregated measures of SES can be conceptualised as properties of areas with an objective of understanding how area constructs relate to health outcomes, or as summaries of individual-level variables documenting the ecologic expression of individual-level associations (Diez Roux 2001). These early ecologic studies were unable to directly assess if spatial variation in health was due to environmental features, or variation in the characteristics of individuals residing in different areas. This resulted in efforts to distinguish between area- and individual-level effects, supported by the use of multilevel models including both area- and individual-level predictors simultaneously in regression models with individuals as the unit of analysis (Diez Roux 2001). The majority of multilevel studies have reported independent effects of area-level SES on health outcomes including cardiometabolic risk, after accounting for individual-level SES (Chaix 2009; Diez Roux 2001; Leal & Chaix 2011).

Area-level SES has also demonstrated associations with the behavioural antecedents of cardiometabolic risk. Smoking and smoking cessation (over six years) are associated with area-level SES after accounting for individual-level factors (Duncan, Jones & Moon 1999; Giskes et al. 2006b). Physical activity level is negatively associated with area-level SES after adjusting for

individual demographic factors (Giles-Corti & Donovan 2002; Kavanagh, AM et al. 2005) and decline in physical activity level (over 9 years) is associated with residing in a poverty area (Yen & Kaplan 1998).

Evidence for associations between area-level SES and individual diet is more equivocal. One study using multilevel models that included individual-level factors, found residents of low-SES areas were less likely than residents of high SES areas to purchase foods that were high in fibre and low in fat, salt and sugar; and they purchased a smaller variety of fruits. There was no association between area-level SES and vegetable variety (Turrell et al. 2009). A separate study of adult women found lower fruit and vegetable consumption, and more fast-food consumption were associated with area deprivation, after adjusting for individual demographic and socioeconomic factors (Thornton, Crawford & Ball 2010). Other studies, however, have found no associations between area-level SES and food purchasing and diet intake after adjusting for individual-level factors (Giskes et al. 2006a; Turrell et al. 2004). Though evidence for associations between area-level SES and individual diet may be equivocal, area-level SES is now well-established as related to poorer health outcomes including cardiometabolic risk (Black, Moon & Baird 2014; Chaix 2009; Diez Roux & Mair 2010; Leal & Chaix 2011).

Area-level SES also covaries with built environment features associated with individual health behaviours and health outcomes. The review by Black and colleagues (2014) reported a consensus across previous reviews that residents in low income or ethnic minority areas in the US have lesser access to healthful foods, and greater access to unhealthful foods than residents of more affluent areas. Though the evidence for disparities in healthful food access in Australia, Canada, and the UK is more equivocal, international evidence suggests disparities in fast food access varies with area-level SES, with greater access to fast-food outlets in low SES areas (Black, Moon & Baird 2014). Similarly, area-level SES is reportedly positively associated with sense of community, residential density, area aesthetics and greenery, traffic safety, and inversely associated with traffic load, and crime (Cerin, Leslie & Owen 2009).

As area-level SES can covary with other environmental conditions (Black, Moon & Baird 2014; Cerin, Leslie & Owen 2009; Chaix, Leal & Evans 2010), aggregated SES measures may also function as proxies for specific features of local environments other than area-level SES,

limiting the causal interpretation of observed associations (Mujahid et al. 2007). It is therefore necessary to assess the specific causal mechanisms through which local environments influence health outcomes, requiring the study of specific environmental features beyond area-level SES (Mujahid et al. 2007). For studies planning to untangle the effects of specific environmental features on health outcomes, it is recommended that area-level SES be included as a covariate intended to reduce area-level confounding, rather than as the predictor of interest (Chaix 2009; Chaix, Leal & Evans 2010).

4.2 Composition beyond area-level socioeconomic status

In research regarding the influences of environmental risk conditions on health outcomes, the term *context* has commonly been used to refer to *neighbourhood effects* (area or group effects) that are independent of individual-level effects (Diez Roux 2001). As noted in the previous section, analysis models (e.g., multilevel models) including both area- and individual-level SES have attempted to explicate the relative effects of each.

Under this definition, the remaining influence of area-level SES on health outcomes as estimated in statistical models after adjustment for individual-level SES is often referred to as *contextual*, while the variance accounted for by individual-level factors is considered the *compositional* effect (Diez Roux 2001; Ross, CE & Mirowsky 2008). This remaining influence (after including individual-level SES) may be due to the influence of other environmental factors that covary with area-level SES (as discussed in the previous section). However, this remaining influence may also reflect the *collective influence* of individuals *beyond* the sum of their individual attributes including SES (Ross, CE & Mirowsky 2008). That is, the local *composition* of individuals (collective of individuals; area-level influence) may have an effect beyond that accounted for by their individual characteristics (individual-level influence). Composition, as more than the sum of individual influences, has not typically been clearly acknowledged in research on environments and health.

The more recent definition of composition and context used within this dissertation (Daniel et al. 2011) has a slightly different conception of composition and context to that described earlier in this section. *Compositional* influences, or the influence of features pertaining to collectives of individuals, are distinct from *individual-level* influences on health outcomes. For example, neighbourhood education level may indicate "collective human capital" which differs conceptually from individual-level education (Ross, CE & Mirowsky 2008). Similarly, collective lifestyles and local descriptive norms (collective population features; composition) represent more than the sum of individual-level beliefs and behaviours, providing a social pressure towards particular beliefs and behavioural choices. The influence of collective lifestyles and local descriptive norms clearly differ from that of individual-level behaviours, though collective lifestyles and local descriptive norms may influence the enactment of individual behaviour. Thus compositional effects reflect the influence of the social milieu an individual is exposed to. Local descriptive norms are one possible expression of composition,

as a collective attribute, beyond area-level SES. Other such compositional measures could include social pathologies and collective hopelessness (Daniel et al. 2011).

Composition, beyond area-level SES, has rarely been considered in research regarding environmental influences on health outcomes. Some few studies have assessed associations between residential area ethnic composition and health outcomes. One recent longitudinal study of US immigrants (Chinese and Hispanic) aged 45-84 years reported associations between decreasing co-ethnic concentration over time and increasing BMI amongst Chinese immigrants (Lê-Scherban et al. 2014). A similar pattern was evident amongst Hispanic immigrants though the confidence intervals were wider and the associations did not reach statistical significance. One cross-sectional US study found individuals residing in areas with greater proportions of Blacks had greater odds of being obese compared to residents of communities with smaller proportions of Blacks, after accounting for area-level SES and individual-level sociodemographic characteristics (Boardman et al. 2005). This association became null after the inclusion of area-level obesity prevalence (i.e., local descriptive obesity norm) to analysis models suggesting that differences in neighbourhood obesity prevalence may mediate the relationship between neighbourhood racial composition and greater odds of obesity among residents of Black communities (Boardman et al. 2005).

The contribution of compositional features, beyond a conception of composition as simply individual level, to individual health outcomes would provide further insights to the possible range of environmental influences on individual health outcomes. Improved understanding of the range of environmental influences, and the conditions under which they may function, can lead to improved, targeted intervention strategies. There is a lack of studies regarding compositional features beyond area-level SES in relation to cardiometabolic risk. This dissertation introduces one novel and under-researched group of compositional features, local descriptive norms, to research regarding environmental influences on health outcomes, and assesses the influence of local descriptive health-related norms on change in cardiometabolic risk.

4.3 Contextual features and cardiometabolic risk

Though few studies have assessed composition beyond area-level SES, in relation to health outcomes, this is not true for contextual features. There is a growing body of empirical evidence linking the contextual physical activity environment with physical activity behaviour and cardiometabolic outcomes (Gascon et al. 2016; Kaczynski & Henderson 2007; Leal & Chaix 2011; Sallis et al. 2012). Similarly, the contextual food environment is related to individual dietary behaviour and its consequences such as obesity and cardiometabolic risk (Black, Moon & Baird 2014; Caspi et al. 2012; Giskes et al. 2011; Holsten 2009; Leal & Chaix 2011; Lovasi et al. 2009).

Contextual physical activity and food environment features are theorised to influence individual-level cardiometabolic outcomes through either the physical activity or dietary behaviour of the individual, respectively. Due to the proposed differences in mechanism underpinning associations with cardiometabolic health, the physical activity and food environments are examined separately within this dissertation. There is, however, some overlap. In particular, the consideration of the local descriptive norm related to body weight, which pertains to both physical activity and dietary behaviour. The following sections review the literature on contextual built environment features related to the 'physical activity' and 'food' environments, relevant to this thesis.

4.3.1 The contextual physical activity environment and cardiometabolic risk

Sallis and colleagues (2006) developed an ecological model (Figure 4.1) built around four domains of active living, where "active living" is a concept incorporating exercise, recreational activities, household and occupational activities, and active transportation. Intrapersonal factors are represented in the centre of the model. Individual-level perceptions of environments are distinguished from objective aspects of environments though both are considered potentially important influences on physical activity (Sallis et al. 2006). Perceived measures reflect an individuals' interaction with the environment and may be influenced by individual (and other) factors (Gebel, Bauman & Owen 2009). Correspondence between objective and perceived measures of environmental features is low (Boehmer et al. 2006; Gebel, Bauman & Owen 2009). Though the influence of perceived environmental factors is important in understanding individual health outcomes, they are not the focus of this dissertation and will, therefore, not be discussed further.

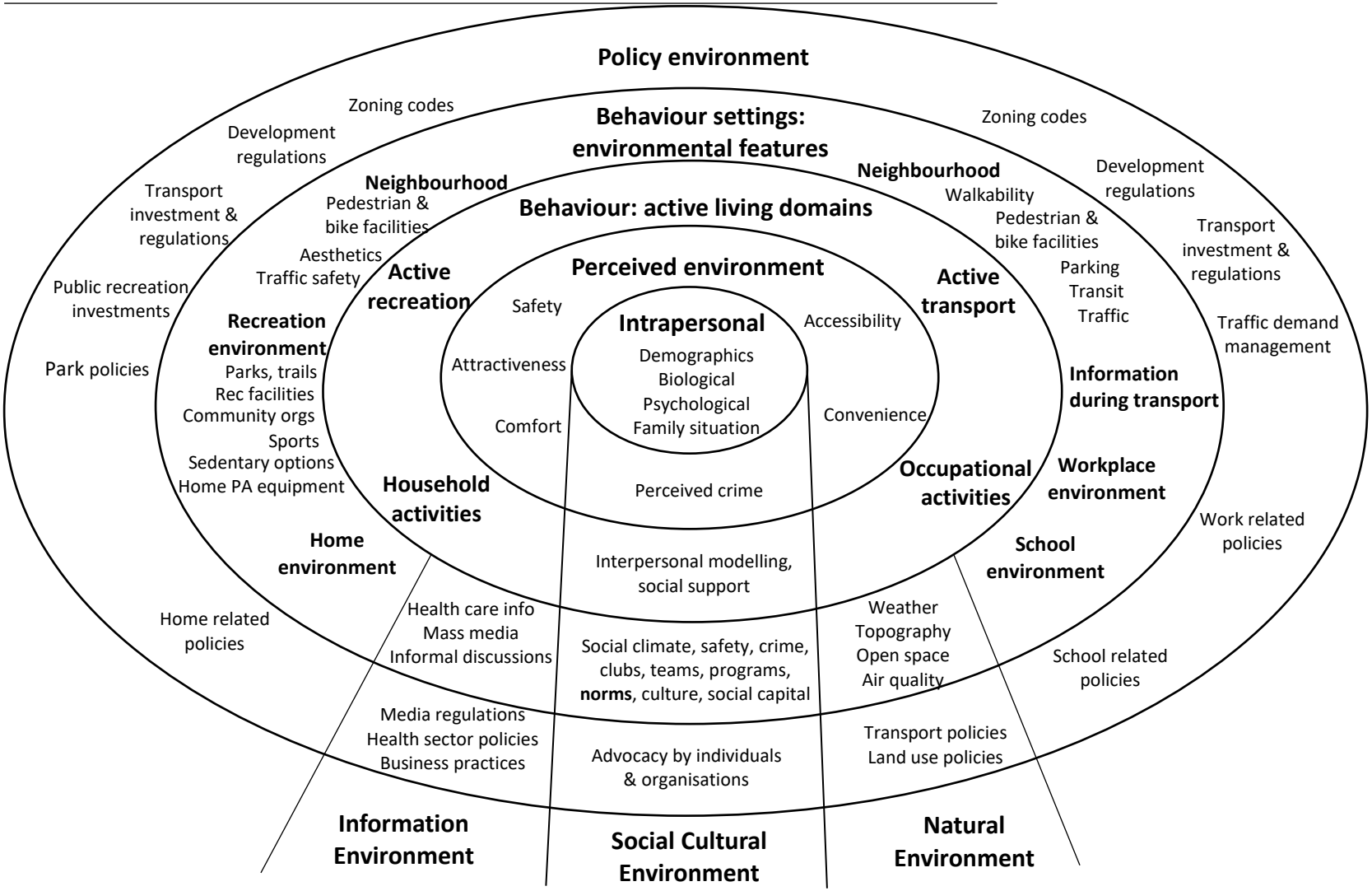


Figure 4.1 Ecological model of four domains of active living (Adapted from Sallis et al. 2006)

The four active living domains of the model by Sallis and colleagues (2006) are likely influenced by different policy and environmental factors. These different influences are outlined in the next layers in the model "behaviour settings" and "policy environment". Behaviour settings are where physical activity may occur. It may be important to consider access and availability of behavioural setting, as well as specific characteristics of the settings (e.g., aesthetics) (Sallis et al. 2006).

The neighbourhood is identified as an important local-area behaviour setting for both active transport and active recreation, though environmental features of importance may differ between these domains. For example, access to and availability of POS may be important for active recreation while walkability may be more important for active transport behaviour.

The policy environment can shape individual or multiple domains of active living through its influence on the built environment, incentives and programs (Sallis et al. 2006). The social and cultural environment, including norms, are shown as cutting across the other levels of the model, as are the natural environment and information environments. These are not limited to specific behavioural domains (Sallis et al. 2006). This dissertation, specifically Study 2, focuses on features of the neighbourhood physical activity environment, including norms defined within the local area, and their influence on change in cardiometabolic risk. It is assumed that much of the influence of the physical activity environment on health is due to the physical activity behaviour of the individual.

Reviews of the physical activity literature have reported associations between several physical activity environment features and physical activity behaviour. Reviews have concluded there are mixed to fairly consistent positive associations between at least one type of physical activity (such as transportation or recreational walking, cycling) or general physical activity, and environmental features including: access to, and availability and variety of, facilities and destinations (e.g., parks and recreational areas, schools, shops and commercial areas); safety (e.g., crime rate, graffiti, incivilities, traffic safety); aesthetics (e.g., greenness, landscaping, upkeep, cleanliness, rated attractiveness); and walkability factors such as mixed land use, residential or population density, street connectivity, and pedestrian infrastructure (e.g., presence and quality of sidewalks, lighting, shade, street furniture) (Bauman et al. 2012; Grasser et al. 2013; Humpel, Owen & Leslie 2002; Kaczynski & Henderson 2007; Koohsari,

Badland & Giles-Corti 2013; McCormack & Shiell 2011; Saelens & Handy 2008; Sallis et al. 2012; Van Cauwenberg et al. 2011).

Fewer reviews have assessed the evidence for associations between environmental features and cardiometabolic outcomes, but of such reviews it seems there is evidence for associations between the physical activity environment and cardiometabolic outcomes (Gascon et al. 2016; Lachowycz & Jones 2011; Leal & Chaix 2011; van den Berg et al. 2015). Leal and Chaix (2011) concluded there were reasonably consistent associations between the absence of parks and greenspaces and increased obesity, and between walkability and lesser obesity. Both walkability and POS, including parks or greenspaces, are of particular interest to this dissertation. Each of these contextual features has been previously linked to both physical activity and cardiometabolic outcomes.

4.3.1.1 Walkability

Walking is the most commonly reported physical activity (Eyler et al. 2003). As walking most often occurs along neighbourhood streets (Eyler et al. 2003), land use patterns and street design may be important influences on walking (and cycling) behaviour (Lovasi, Grady & Rundle 2012; Pikora et al. 2003; Sallis et al. 2004). Various features of local environments have been theorised to be supportive of local walking (and cycling) behaviours including residential density, land use diversity, well-connected transportation networks, destinations (e.g., shops and services, public art, cafes, POS), traffic (noise, volume, speed and safety), signs of physical disorder (e.g., litter, graffiti and crime), sense of safety, presence and quality of sidewalks, lighting, trees, greenery, and natural scenery (Lovasi, Grady & Rundle 2012; Sugiyama et al. 2012).

Within the range of built environment features believed to be supportive of walking, two concepts, connectivity and proximity, have been widely used to express walkability (Coffee 2005; Frank, Andresen & Schmid 2004; Lovasi, Grady & Rundle 2012). Connectivity is operationalised by intersection density, and proximity as population or dwelling density, land use mix and net retail area (retail space footprint ratio to car parking area) (Coffee 2005; Coffee et al. 2013; Frank et al. 2010; Frank et al. 2005; Leslie et al. 2007). Studies within the International Physical Activity and the Environment Network (IPEN) have consistently used this definition of walkability.

Components of connectivity and proximity can be considered individually, or combined to form environmental 'walkability' scores. Walkability information can also be collected and expressed in other ways. Examples include individual-level perceived walkability (e.g., Cerin et al. 2008; Cerin et al. 2006) and data collected using a 'walking audit instrument' (e.g., Cain et al. 2014). Individual-level perceptions of local environments can be useful, but, as already stated, perceptions typically differ from objective measures and are not the focus of this dissertation.

'Walking audit instruments' involve the assessment of features, such as building height, block length and street and sidewalk width, usually performed by trained auditors (Ewing, R & Handy 2009). Collecting data using such field audits can be labour intensive, time consuming and expensive (Clifton, Livi Smith & Rodriguez 2007). Data may be specific to the time period within which they were collected. That is, historical data are unavailable through direct field audit (unless an audit was previously conducted at the relevant time point).

Walkability scores can be constructed using GIS for previous time-points, if appropriate data are available. Many studies have investigated associations between walkability scores (based on proximity and connectivity) and physical activity behaviour, particularly walking. These studies often report positive associations between walkability and walking behaviour (e.g., Carlson et al. 2012; Frank et al. 2006; King et al. 2011; Owen et al. 2007; Sundquist et al. 2011; Van Dyck et al. 2010) and review articles generally concede there is a reasonable body of evidence supporting associations between walkability and walking, particularly walking for transport (Hajna et al. 2015; Lovasi, Grady & Rundle 2012; Saelens & Handy 2008; Sallis et al. 2012).

As physical activity, including walking, is positively associated with improved cardiometabolic outcomes, it follows that greater walkability may be inversely associated with cardiometabolic risk. Inverse associations have been reported between objectively measured walkability (or its components) and cardiometabolic risk (Coffee et al. 2013), obesity (e.g., Frank, Andresen & Schmid 2004; Grafova et al. 2008; Sallis et al. 2009), and diabetes incidence (Booth et al. 2013). However, many studies have reported null findings between walkability and cardiometabolic outcomes including weight status (e.g., Berry et al. 2010; Christian et al. 2011). Regardless, most (Leal & Chaix 2011; Lovasi, Grady & Rundle 2012) but not all (e.g.,

Durand et al. 2011) reviews have concluded that there is fairly consistent evidence for inverse associations between walkability and cardiometabolic risk (Leal & Chaix 2011; Lovasi, Grady & Rundle 2012).

4.3.1.2 Public open space

Another important contextual physical activity environment feature is POS. In place and health research, POS is mainly conceptualised as parks and greenspaces, with less focus on other types of public open spaces (e.g., public plazas, nature reserves, greenways) (Koohsari et al. 2015a). However, different studies, particularly from different fields, use varying definitions (for discussion see Koohsari et al. 2015a). Regardless of differences in POS definitions, the accessibility and availability of POS offers opportunities for recreation and physical activity for little to no cost to the individual (Hunter et al. 2015). POS can be a space within which people engage in physical activity, a destination to which people actively travel, and can be used as part of an active transit route or as part of a recreational walk or running route (Koohsari et al. 2015a). By supporting active lifestyles, POS may reduce cardiometabolic risk (Astell-Burt, Feng & Kolt 2014).

Methods of expressing POS differ between studies. Commonly used methods include access (proximity) and availability (count, area and density) of POS, and a Normalised Difference Vegetation Index (NDVI) representing 'greenness', though other methods have also been used such as audits by trained assessors (e.g., Ellaway, Macintyre & Bonnefoy 2005). Access has been expressed as the distance, either Euclidean or network, to the nearest POS from residential address (e.g., Tamosiunas et al. 2014). Availability may be expressed as a count of POS within a spatial unit, or the proportion of a spatial unit that is defined as POS (e.g., Mitchell & Popham 2008; Richardson & Mitchell 2010). An NDVI provides an indication of vegetation photosynthetic activity and is calculated from satellite imagery as the difference between the near-infrared and the visible reflectance of the vegetation divided by the sum of the two (Defries & Townshend 1994). Studies regarding POS may focus on particular attributes of POS, such as the type (e.g., Giles-Corti et al. 2005; Jones, Hillsdon & Coombes 2009; Paquet et al. 2013), size (e.g., Giles-Corti et al. 2005; Paquet et al. 2013), quality (e.g., Giles-Corti et al. 2005; Hillsdon et al. 2006; Paquet et al. 2013), or available facilities (e.g., Giles-Corti et al. 2005). The many differences in the definition and operationalisation of POS makes comparisons of

findings, such as associations with physical activity or cardiometabolic risk, between studies difficult (Lachowycz & Jones 2011).

Over the last decade, the number of studies examining the influence of POS on physical activity has increased (Koohsari et al. 2015a). The review by Kaczynski and Henderson (2008) concluded POS was generally positively related to physical activity. However, a more recent review by Lachowycz and Jones (2011) concluded that evidence for associations between access and availability of POS, and physical activity was mixed. Though the majority (66%) of studies reported a positive association, only 40% found an association that appeared unambiguous (Lachowycz & Jones 2011). Some studies have reported negative associations (e.g., Maas et al. 2008; Troped et al. 2010).

The inconsistent findings may relate to contextual or cultural factors, and the diverse ways used to conceptualise and operationalise POS (Koohsari et al. 2015a). Kaczynski and colleagues (2009) reported no association between distance to closest POS and moderate to vigorous physical activity, though greater total park area within 1km of participants place of residence was associated with increased odds of meeting physical activity recommendations.

POS is also related to cardiometabolic risk factors and disease, though not always consistently. The Leal and Chaix (2011) review concluded there were reasonably consistent associations between an absence of POS and obesity, with five of seven studies reporting associations. The review by Lachowycz and Jones (2011) concluded the majority of studies reported some evidence of an association between POS and BMI, but mixed results across subgroups and POS measures. Studies assessing associations between POS and cardiometabolic outcomes other than weight status are less common. However, a recent review concluded there was sufficient evidence to support a reduction in CVD mortality risk for individuals residing in greener areas (Gascon et al. 2016).

There is a general consensus that contextual environmental features such as walkability and POS are associated with physical activity and cardiometabolic outcomes, but, further research is needed to understand the inconsistent findings. Additionally, researchers need to extend the outcome measure to include cardiometabolic risk beyond the predominant focus on weight status. Though the differences in reported findings may be attributable to differences in walkability and POS measures, these differences may also reflect the complexity of the

relationship between compositional and contextual environmental features, including the potential for interaction effects between environmental features in relation to health outcomes.

4.3.2 The contextual food environment and cardiometabolic risk

Glanz and colleagues (2005) proposed a conceptual model (Figure 4.2) for the study of food environments. Based on an ecological framework, the model incorporates constructs reported or hypothesised to be related to healthful eating patterns.

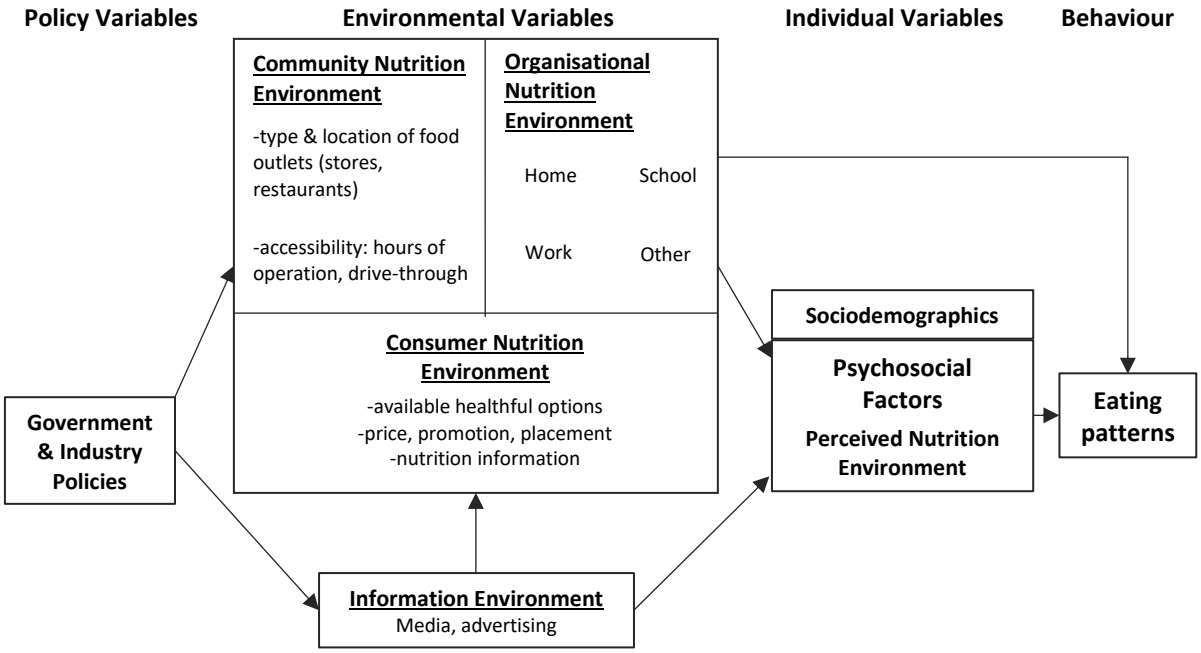


Figure 4.2 Conceptual model of the influence of local food environments on dietary behaviour (Adapted from Glanz et al. 2005)

The model identifies four types of food environments, each affected by policies of governments and other organisations (Glanz et al. 2005). There are two pathways of environmental influence, direct through the provision of access to resources, and indirect whereby environmental effects are moderated or mediated by demographic, psychosocial, or perceived environmental variables. The *community nutrition environment* focuses on the distribution of food resources, that is, type, location and accessibility (including drive through windows and hours of opening) of food outlets. Other food sources such as homes and cafeterias in schools, workplaces or other locations are termed *organisational nutrition environments*. The *consumer environment* reflects what consumers encounter within and around a retail food outlet such as food types available, price, promotions, placement, range

of choices, quality and freshness, and nutritional information. Lastly, the *information environment* refers to media reports and advertising, affected by government and industry policies, which could affect individual attitudes and the appeal of certain foods and food sources. The information environment can operate on a national, regional, neighbourhood, store or restaurant level. Though the focus of this dissertation is on the community nutrition environment, particularly, contextual built environment features relating to the availability of fast and healthful food, this is not to discount the importance of the organisational and consumer nutrition environments. A focus on the community nutrition environment aligns with the conceptual framework underlying this thesis (Figure 3.3) while enabling matched compositional and contextual features to be expressed within the same spatial scale.

Reviews articles conclude that the majority of food environment research has focused on the community nutrition environment (Black, Moon & Baird 2014; Caspi et al. 2012). GISs are commonly used to determine the geospatial location of food sources (e.g., supermarkets, green grocers, restaurants and fast-food outlets) enabling measurement of their accessibility, availability and, less commonly, diversity (Black, Moon & Baird 2014; Charreire et al. 2010). Accessibility measures the distance between food resources and an individual's place of residence (or other location of interest) using Euclidean distance, road-network distance or travel time (Black, Moon & Baird 2014). Availability is the density of different types of food outlets within a spatial unit, expressed as a total count, count per population, per square area, or kernel density estimation (Black, Moon & Baird 2014). Diversity measures the different types of outlets for a category, for example, the number of different types of fast food outlets representing choice of fast foods (Black, Moon & Baird 2014).

Food outlets are commonly classified as either healthful or unhealthful. Supermarkets tend to offer a variety of healthful foods at low cost (Bodor et al. 2008; Glanz et al. 2007). However, supermarkets also offer a range of unhealthful food options (Caspi et al. 2012). Convenience stores sell mostly pre-prepared, calorie dense food and only a small amount of fresh produce, typically at high prices (Lovasi et al. 2009; Zenk et al. 2005). Fast-food outlets sell energy dense foods high in salt, fat and sugar with few healthier options available (Bowman & Vinyard 2004; Prentice & Jebb 2003; Rosenheck 2008; Stender, Dyerberg & Astrup 2007). Full-service restaurants offer a wider range of menu options and are consequently difficult to classify, though generally tend towards unhealthful opportunities (Saelens et al. 2007).

Associations between the community food environment and cardiometabolic outcomes have been reported. Reviews have documented reasonably consistent associations between accessibility to supermarkets and healthier diets and lower body weight (Larson, Story & Nelson 2009; Leal & Chaix 2011). Conversely, greater accessibility to convenience stores and fast-food outlets is associated with poorer diets and higher body weight (Larson, Story & Nelson 2009; Leal & Chaix 2011). Studies of other cardiometabolic risk factors have observed associations between a higher availability of healthful food and reduced likelihood of hypertension (Mujahid et al. 2008), and lower type 2 diabetes incidence over 5 years (Auchincloss et al. 2009). Fast-food outlet availability has been associated with detrimental change in blood pressure over 1 year in neighbourhoods with low walkability (Li et al. 2009), and mortality and hospital admissions for acute coronary syndromes (Alter & Eny 2005) in models adjusted for individual and area-level covariates.

Many studies, however, have found no associations between aspects of the food environment and cardiometabolic outcomes. For example, one study found no association between the presence of food stores (supermarkets, grocery and convenience stores) and prevalence of diabetes and high cholesterol (Morland, Diez Roux & Wing 2006). Similarly, a study of low-income women in five US states found no associations between food availability (density of grocery stores, fast-food outlets, restaurants, or minimarts) and BMI or CVD risk (Mobley et al. 2006).

It is generally accepted that the contextual food environment (e.g., availability of fast and healthful food resources) is associated with individual dietary behaviour and consequent health outcomes, typically weight status. Multiple environmental features likely act simultaneously on individual-level health outcomes. Moreover, environmental features may interactively influence individual-level health outcomes. Interactive effects may partly underlie differences in reported findings between studies.

4.4 Interactions between environmental features in relation to cardiometabolic risk

Several reviews have called for research to include assessment of interactive effects between environmental features in relation to health outcomes (e.g., Leal & Chaix 2011; Lovasi, Grady & Rundle 2012; Schüle & Bolte 2015). Despite this, few studies report assessing such interactive effects. The 2011 review by Leal and Chaix on associations between environmental features and cardiometabolic risk factors included a total of 131 papers of which seven papers (one on blood pressure, and six on obesity) reported assessing interactions between environmental features (Leal & Chaix 2011). A more recent review regarding associations between environmental features and health outcomes focused exclusively on multilevel studies (Schüle & Bolte 2015). Of the 33 papers reviewed, six assessed interactions between environmental features of which two assessed predictors of physical activity, one paper regarded smoking behaviour, and three papers focused on birth-weight as the outcome (Schüle & Bolte 2015). Though there may have been an increase in the proportion of studies considering interactive environmental effects on health outcomes, more studies are needed to further develop understanding of the complex relations between environments and health outcomes.

Of studies regarding interactions between environmental features in relation to cardiometabolic risk, a US study of older adults reported interaction effects such that the influence of greater fast-food outlet density on increasing blood pressure levels was reduced amongst individuals residing in highly walkable neighbourhoods (Li et al. 2009). One UK study reported that associations between area-deprivation and mortality from circulatory diseases were lower in populations residing in the greenest areas (Mitchell & Popham 2008). A study of adults in the US reported an interaction between walkability and area-level income such that the magnitude of the association between walkability and transport walking was greater in high-income areas (Sallis et al. 2009). A Belgian study of adolescent (13-15 years) physical activity behaviour reported neighbourhood walkability was positively associated with accelerometer measured physical activity (both average activity level and mean minutes of moderate to vigorous activity per day) only in low-SES neighbourhoods (De Meester et al. 2012).

Conversely, a study of Swedish adults reported no interaction effects between neighbourhood walkability and area-level deprivation in relation to 3-year type 2 diabetes incidence

(Sundquist et al. 2015). Similarly, a cross-sectional multiethnic UK study of adults reported no interactions between fast-food outlet availability (count) and urban/rural location, or fast-food outlet availability and area-level social deprivation score in relation to type 2 diabetes (Bodicoat et al. 2015).

No consistent findings have emerged from the few studies regarding environmental interaction effects in relation to cardiometabolic outcomes. No studies have assessed interactions between local descriptive norms and built environment features in relation to cardiometabolic outcomes. This is an area that requires more research and this dissertation helps to address this research gap.

4.5 Critical appraisal of the literature

Though a body of evidence reports associations between environmental risk conditions and cardiometabolic outcomes independently of individual-level factors, there are a number of research gaps. Moreover, a number of methodological limitations commonly found within studies of this field which must be acknowledged.

Individuals live in areas that consist of numerous potential risk conditions acting upon health simultaneously. Studies investigating the effects of different environmental exposures often do not report analyses considering these variables simultaneously in regression models (Leal & Chaix 2011). Models that include both contextual and compositional features simultaneously may assist in understanding the unique contribution of each component to the development of cardiometabolic risk. Furthermore, environmental features are likely to interact (see Sections 3.5 and 4.4), where some features, for example local descriptive norms, may indirectly act as predisposing factors, while other features such as built environment context, may function as enabling factors. Relatively few studies have assessed interactions between environmental features and health outcomes.

A key limitation of the current body of research is that the majority of studies assessing the influence of environmental features in relation to health outcomes and behaviours use self-reported outcome measures (Koohsari et al. 2015b; Leal & Chaix 2011; Mackenbach et al. 2014; Rahmanian et al. 2014). Though self-report is a practical, low cost method for collecting information in large samples (Gorber et al. 2007), such information is not always accurate (Brenner & DeLamater 2014; Miller et al. 2008). In collecting self-report information, questions may be misunderstood, recall can be inaccurate, and social desirability may introduce biases (Brenner & DeLamater 2014; Gorber et al. 2007; Miller et al. 2008). Associations may be distorted by the use of self-report measures and it has been recommended that measured outcomes be used for future research (Leal & Chaix 2011).

A further concern is causal direction as the majority of studies investigating associations between environments and cardiometabolic outcomes rely on a cross-sectional design (Leal & Chaix 2011; Mackenbach et al. 2014; Malambo et al. 2016; Schüle & Bolte 2015). Cross-sectional studies limit inference on the direction of causation (Diez Roux & Mair 2010; Leal & Chaix 2011) and reverse causation is a possible explanation of any associations found. Poor

health may be a cause, rather than a result, of place of residence (Yen, Michael & Perdue 2009). Temporal order supports causal interpretation (Daniel, Moore & Kestens 2008). Temporality can only be assessed using a longitudinal design, with exposure occurring before the outcome measure. Longitudinal studies are therefore needed to provide evidence to support causal relationships between environmental features and cardiometabolic outcomes.

Lastly, the choice of spatial unit used to express environmental exposures is a concern. Administrative data with spatial identifiers, such as census data for area-level SES, have often been used, especially in older research regarding environmental risk conditions and health. Consequently, the majority of studies investigating associations between environments and cardiometabolic outcomes use predetermined administrative spatial units (Leal & Chaix 2011). Predetermined administrative spatial units are useful due to ease of linkage with, for example, census data (Riva et al. 2009). However, environmental features may not be optimally defined and expressed within these spatial units (Riva et al. 2009). There is a growing recognition that the choice of spatial unit influences study results in health research (Chaix et al. 2009; Coffee 2013; Coffee et al. 2013; Cummins et al. 2007; Gauvin et al. 2007). It is now widely recognised that expressing environmental measures for predetermined administrative spatial units may not validly reflect the environmental exposures of individuals (Chaix et al. 2009).

When expressing area-level exposures, the choice of definition of area boundaries influences between-area variation and consequent estimation of the effect of area-level exposures on health outcomes (Coffee et al. 2013; Flowerdew, Manley & Sabel 2008; Riva et al. 2009). This issue is known as the modifiable areal unit problem (MAUP). The MAUP includes two aspects, the effect of scale, and of zoning (Openshaw 1984; Openshaw & Taylor 1979). The size of the spatial unit used (scale) can result in differences in statistical associations (Coffee et al. 2013; Flowerdew, Manley & Sabel 2008; Openshaw 1984). Similarly, different spatial configurations, that is, how the study area is subdivided (zonation), even at the same scale, can result in different statistical findings (Coffee et al. 2013; Flowerdew, Manley & Sabel 2008; Openshaw 1984).

The MAUP is of particular concern when using predetermined administrative spatial units. Predetermined administrative spatial units are often arbitrarily determined, and can be aggregated to form different spatial arrangements (zonation) of differing sizes (scale)

(Fotheringham & Wong 1991; Gauvin et al. 2007; Jelinski & Wu 1996). Additionally, the use of predetermined administrative spatial units does not take into account where the participant resides within the spatial unit. If the participant lives close to the border of a predetermined administrative spatial unit, the spatial unit may not reflect the individuals' environmental exposures.

It is recommended that an ego-centred definition of area be used, that is, spatial units centred on individuals' residences (Chaix et al. 2009). For example, circular buffers can be constructed around the individual's place of residence. Ego-centred buffers can be further refined by defining in terms of distance along the road network (Chaix et al. 2009). Buffers designed based on distance along the road-network will vary in size and shape but can allow researchers to account for barriers in accessibility (Chaix et al. 2009).

The use of buffers at set distances (standardised between studies) to operationalise an individual's residential neighbourhood has helped to manage the MAUP, though the most appropriate buffer size is still open to debate (Brownson et al. 2009). The most appropriate scale of spatial unit may be specific to both the outcome of interest and the proposed environmental influence (Brownson et al. 2009; Koohsari, Badland & Giles-Corti 2013). It is therefore recommended to define these road-network buffers on different scales, that is, at different road-network distances whenever possible (Brownson et al. 2009; Chaix et al. 2009).

This section has identified an important research gap and key methodological issues commonly found in research assessing associations between environmental exposures and health outcomes. Environmental exposures occur simultaneously though few studies have assessed the influences of multiple environmental exposures simultaneously within regression models. Moreover, environmental exposures may interact in relation to health outcomes though this has rarely been assessed. In addition, the majority of previous studies: 1) have relied on self-report measures to express the outcome though the use of such measures may distort associations between environmental predictors and the outcome measure; 2) are cross-sectional in design, limiting inference on the direction of causality; and 3) have used predefined administrative units though these spatial units may not best reflect an individual's actual exposures and a subject to the MAUP.

This dissertation contributes to advancing knowledge regarding environmental exposures and health outcomes by investigating a novel measure, compositional local descriptive norms, in models simultaneously with contextual built environment features to predict 10-year change in cardiometabolic risk. Local descriptive norms may be important influences on health behaviour and outcomes and may provide a useful lever for improving population health. The research in this dissertation also assesses potential interaction effects between compositional local descriptive norms and contextual built environment features in relation to change in cardiometabolic risk. There is a need to understand under what conditions environmental features influence health outcomes to better inform appropriate intervention strategies. This research attends to the methodological limitations identified above by being longitudinal in design, using a clinical, as opposed to self-report, outcome measure, and expressing environmental exposures within participant-centred road-network buffers. The following section outlines the operational model and associated research hypotheses that were tested to achieve the research aim and objectives.

4.6 Research aim and objectives, operational model, and hypotheses

The conceptual framework underlying this research was presented in Chapter 3 as Figure 3.3. Pathways of interest (circled in red, Figure 4.3) have been extracted from the conceptual framework so that specific associations, representing these pathways, can be empirically tested.

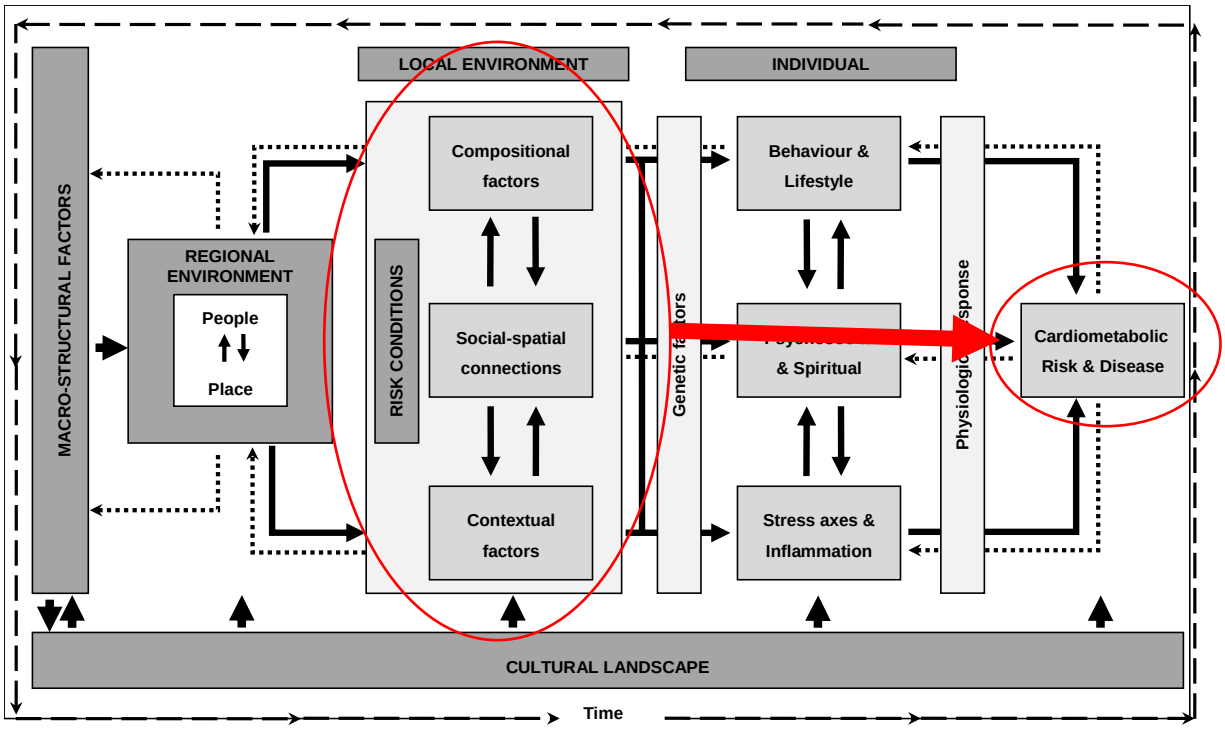


Figure 4.3 Conceptual framework with pathways of interest to this dissertation highlighted (Reproduced from Daniel et al. 2011)

The associations of interest were tested using theoretically cohesive models focused within the physical activity and food environments, with models designed so that compositional and contextual features were 'matched'. Environmental features theorised to be associated with the physical activity environment were included in models assessing the physical activity environment and cardiometabolic risk. The same approach is applied in the construction of the models developed for features representing the food environment.

The aim of this research was to assess the independent and interactive contributions of local descriptive norms and built environment features on 10-year change in individual-level cardiometabolic risk in a population-based, biomedical cohort. The research used 3 waves of individual-level data from the North West Adelaide Health Study (NWAHS), a 10-year biomedical cohort comprised of over 4000 adult participants originally recruited within the northern and western regions of metropolitan Adelaide.

The physical activity environment was expressed as local compositional norms for physical inactivity and overweight/obesity, and contextual walkability and availability of POS. The food environment was expressed as compositional norms for overweight/obesity and unhealthful fruit intake, and contextual availability of healthful and unhealthful food. Details of the methods, including datasets used and operationalisations of measures, are provided in Chapter 5.

The research aim incorporates three objectives (Figure 4.4) and four hypotheses (Table 4.1, next page).

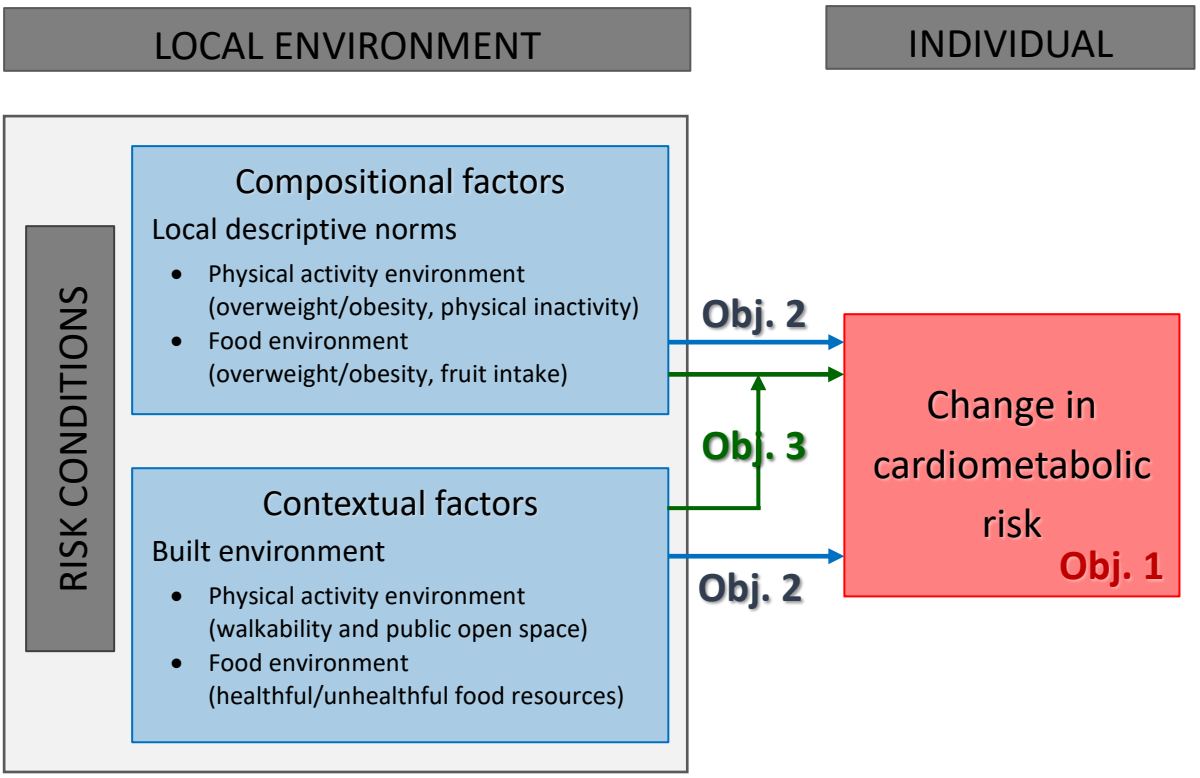


Figure 4.4 Operational model including research objectives

Obj. 1 Validation of a continuous index of cardiometabolic risk constructed from five clinical risk factors, and assessment of its utility in reflecting change in cardiometabolic risk over time;

Obj. 2 Evaluation of the contributions of compositional and contextual features to change in cardiometabolic risk over 10 years; and

Obj. 3 Evaluation of the joint effects of compositional and contextual features to change in cardiometabolic risk.

Table 4.1 Research hypotheses

Objective 1: Validation of a continuous clinical index of cardiometabolic risk (cCICR) constructed from five clinical risk factors, and assessment of its utility in reflecting change in cardiometabolic risk over time	
-	No related hypotheses
Objective 2: Evaluation of the independent contributions of compositional and contextual features to change in cardiometabolic risk over 10 years	
1	Composition: Greater level of adverse local descriptive norm (overweight/obesity, physical inactivity and insufficient fruit intake) will predict increasing cardiometabolic risk over time
2	Context: Greater level of positive feature (walkability, POS availability and healthful food availability) will predict lesser increases in cardiometabolic risk over time
3	Context: Greater level of adverse feature (fast-food availability) will predict increasing cardiometabolic risk over time
Objective 3: Evaluation of the joint effects of compositional and contextual features to change in cardiometabolic risk *Hypothesis 4 (Objective 3) can be expressed in 3 ways:*	
4a	Adverse composition x adverse context: Greater level of adverse local descriptive norm (e.g., overweight/obesity) and greater level of adverse feature (e.g., fast-food availability) will interact to amplify increasing cardiometabolic risk over time
4b	Favourable composition x favourable context: Lesser level of adverse local descriptive norm (e.g., overweight/obesity) and greater level of positive feature (e.g., walkability) will interact to attenuate increasing cardiometabolic risk over time
4c	Favourable composition x adverse context: Lesser level of adverse local descriptive norm (e.g., physical inactivity) will interact with level of adverse feature (e.g., lesser walkability) to attenuate increasing cardiometabolic over time

NB: hypothesised associations independent of other environmental exposures and individual-level covariates

This chapter has presented the current state of the literature regarding environmental influences on cardiometabolic risk and outlined the research gap that this dissertation addresses. The following chapter provides information on the datasets, measures and techniques used to meet the research aim and objectives.

Chapter 5

Methods

5 Methods

This dissertation includes three studies presented as published manuscripts (Chapters 6, 7 and 8) with each manuscript providing details of the methods specific to that study. Due to journal word constraints, the methods sections within each publication provide only limited information on methods and datasets used to construct measures. To aid the understanding of this research, this chapter summarises the overall research design, provides additional detail on the datasets and methods used to operationalise variables, and outlines the analytic approaches used to assess the research objectives.

Section 5.1 outlines the design of the overall project. The study region is introduced in **Section 5.2**. **Section 5.3** provides a brief introduction to GIS. **Section 5.4** describes the datasets used to construct utilised measures and **Section 5.5** describes the participants. Spatial units used to operationalise environmental exposures are defined in **Section 5.6**. **Section 5.7** defines the measures used and their operationalisation. **Section 5.8** outlines the analytic approaches used for each of the studies and **Section 5.9** concludes the chapter.

5.1 Overall project design

The included research is observational in design, incorporating biomedical cohort data linked with spatial datasets through the use of a GIS (Section 5.3). The research used three waves of individual-level data from the NWAHS (Wave 1 [2000-03], Wave 2 [2005-06], and Wave 3 [2008-10]; Section 5.4.1), a 10-year prospective biomedical cohort comprised of over 4000 adult participants originally recruited within the northern and western regions of metropolitan Adelaide (Section 5.2). This research was part of the Place and Metabolic Syndrome (PAMS) Project, funded by a combined National Health and Medical Research Council (NHMRC) Partnership (#570150) and Project (#631917) grants.

The PAMS Project aimed to link individual-level health data with environmental features hypothesised to be associated with cardiometabolic risk. The PAMS Project received ethics approvals (Appendix A) from the University of South Australia Human Research Ethics Committee (HREC) (P029-10 and P030-10), the Central Northern Adelaide Health Service HREC (Application No. 2010010), and the South Australian Department of Health HREC (Protocol No. 354/03/2013). Ethics approval (Appendix B) for use of the South Australian Monitoring and Surveillance System (SAMSS) data, used to operationalise local descriptive norms, was obtained from the South Australian Department of Health HREC (HREC/13/SAH/53). SAMSS is an ongoing surveillance system monitoring trends in risk factors and chronic diseases (Section 5.4.6) (PROS 2012).

5.2 Study region

This project focused on the northern and western regions of Adelaide (Figure 5.1), the capital city of South Australia. These regions accounted for 38% of Adelaide's 1.1 million population and 28% of South Australia's population in 2001 (ABS 2001, 2003). The northern and western regions of Adelaide reflect the demographic profile of the State's population (Grant et al. 2006) and are of particular interest on the basis of elevated cardiometabolic risk relative to other areas (Dal Grande et al. 2004; South Australian Department of Health 2005).

The environmental exposures assessed in Studies 2 and 3 are typically expressed within urban areas and may not be relevant in peri-urban locations characterised by lower population and retail densities. Further, associations between environments, health behaviours and outcomes may differ between urban and rural regions (Wilcox et al. 2000). Studies 2 and 3 therefore limited the investigated region to urban areas only, defined as Census Collection Districts (CDs) with a population density of >200 persons per hectare (ABS 2001).

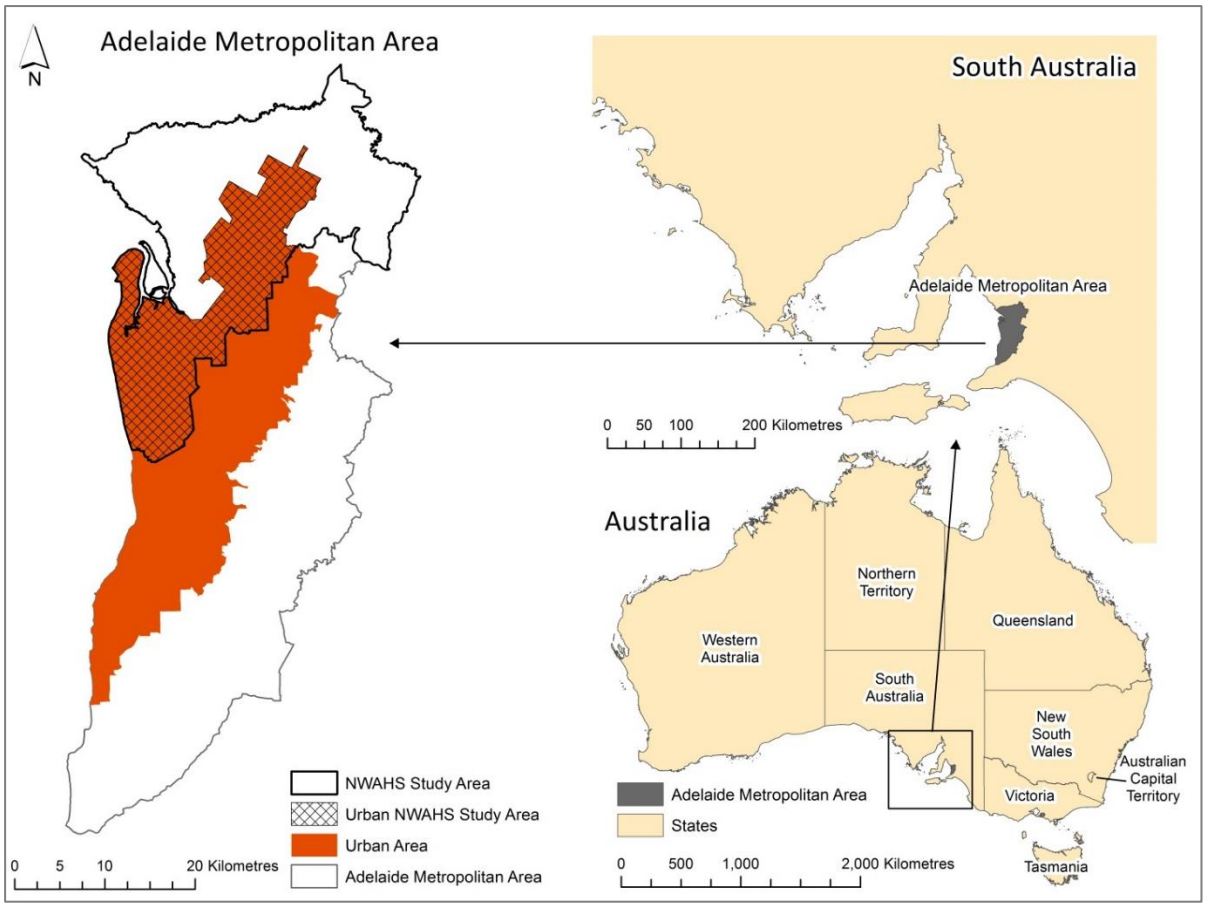


Figure 5.1 Study regions: NWAHS study area, and the urban NWAHS study area used for Studies 2 and 3

5.3 Geographic Information Systems

Data linkage for this project used a GIS. Use of a GIS involves the application of analytic tools that process spatial data into information about a specific geographic portion of Earth (DeMers 2009). A GIS can be conceptualised as a series of data layers (Figure 5.2) with observations within each layer mapped to the Earth's surface via a specific coordinate system (e.g., longitude and latitude; Leslie et al. 2007). Data which include a spatial reference (e.g., longitude and latitude) can be linked with other data layers based on the same reference system.

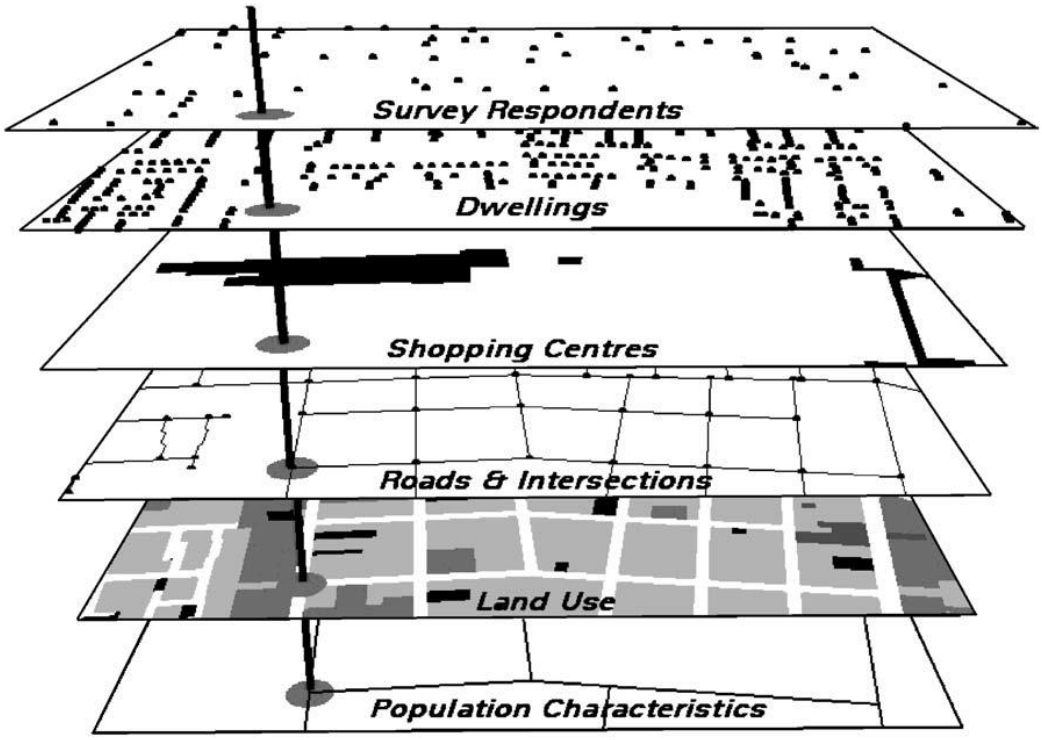


Figure 5.2 Graphical representation of a GIS demonstrating layers of information linked through spatial reference information
(Reprinted from Leslie et al. 2007 with permission from Elsevier)

5.4 Data sources

The three studies included in this dissertation used different methods and measures, drawing on a variety of data sources. All three studies used NWAHS data to represent individual-level information (health outcomes, sociodemographic and behavioural covariates). Study 1, addressing Objective 1, uses only the NWAHS data. For Studies 2 and 3, attending Objectives 2 and 3, NWAHS data were spatially linked with other datasets which were used to construct environmental exposure measures representing the physical activity and food environments. Compositional measures were local descriptive norms for overweight/obesity, physical inactivity, and insufficient fruit intake, and area-level SES (area-level covariate). Contextual built environment features included walkability (four component index), availability of POS, availability of fast-food outlets, and availability of healthful food resources.

Compositional and contextual information were extracted from a range of data sources (Table 5.1). To enable spatial data linkage, datasets needed to include spatial identifiers (georeferences). These data sources are described in the following subsections (5.4.1 to 5.4.7). Details of the operationalisation of measures constructed from these datasets are provided in Section 5.7.

Georeferenced SAMSS data, used to operationalise local descriptive norms, were only available from 2006 onwards. One objective of this research was to assess the contributions of compositional and contextual features simultaneously in analysis models (i.e., their independent effects). This would best be performed by defining these exposures as contemporaneously as was practicable. Therefore, all environmental exposures were defined at time points as close as possible to the NWAHS Wave 2 (2005-2006) participant data (Table 5.1). In Studies 2 and 3, participants who moved between Waves 1 and 2 were excluded from analyses.

Table 5.1 Database sources for individual-level and area-level information used in Studies 1-3

Measures	Database	Year	Source
Individual-level data (Studies 1-3)			
S1: CVD, T2DM, clinical cardiometabolic risk data, age	North West Adelaide Health Study	2000-2010	SA Health[1]/The University of Adelaide
S2 & S3: outcome (cardiometabolic risk as HbA_{1c}); sociodemographic and behavioural covariates		W1: 2000-2003 W2: 2004-2006 W3: 2008-2010	
Contextual features (Studies 2 and 3)			
S2: Physical activity resources: public open space (POS)	Validated Adelaide POS dataset	2007	Spatial Epidemiology and Evaluation Research Group, School of Health Sciences, University of South Australia
S2: Walkability[2]	South Australian Property Cadastre	2007	Department of Planning, Transport and Infrastructure[1]
	South Australian Retail Database	2007	Department of Planning, Transport and Infrastructure[1]
	Adelaide StreetPro™ Road Data	2006	Pitney Bowes Business Insight
S3: Food resources: fast-food and healthful food availability	South Australian Retail Database	2007	Department of Planning, Transport and Infrastructure[1]
Compositional features (Studies 2 and 3)			
Health-related norms: Overweight/obesity (S2 & 3), physical activity (S2), diet (S3)	South Australian Monitoring and Surveillance System (SAMSS)	2006-2010	SA Health[1]/The University of Adelaide
Area-level covariates (Studies 2 and 3)			
Area-level socioeconomic status	Population and Housing Census	2006	Australian Bureau of Statistics

Abbreviations: CVD – cardiovascular disease; S1 – Study 1; S2 – Study 2; S3 – Study 3; T2DM – type 2 diabetes; W1 – Wave 1; W2 – Wave 2; W3 – Wave 3

[1] South Australian Government department; [2] For Walkability, data were sourced from those listed to construct component variables

5.4.1 North West Adelaide Health Study

The NWAHS provided individual-level health, sociodemographic and behavioural information used in each of the studies. Individual-level health information included: self-report of doctor-diagnosed cardiometabolic disease (type 2 diabetes, heart attack, stroke, angina, or transient ischaemic attack/mini stroke); and clinically measured cardiometabolic risk factors (blood pressure; height and weight [to calculate BMI]; waist and hip girths; and a fasting blood sample used to determine triglyceride, total cholesterol, high density lipoprotein [HDL], glucose, and HbA_{1c} concentrations). Self-reported sociodemographic and behavioural information used in the studies included: age; sex; employment status; education level; marital status; and smoking status.

The NWAHS is a longitudinal cohort investigating the prevalence of chronic conditions, including diabetes and cardiovascular disease, and associated risk factors such as obesity, smoking, alcohol consumption, physical inactivity, high blood pressure and high cholesterol (Grant et al. 2006). Data include biomedical information collected during clinic visits, and self-reported information collected via Computer Assisted Telephone Interview (CATI) and paper-based questionnaires. The NWAHS includes three waves of data collection: Wave 1 (2000-03), Wave 2 (2005-06), and Wave 3 (2008-10).

At baseline, the NWAHS comprised 4056 randomly selected men and women aged 18 years and older. Households in the northern and western areas of Adelaide, defined by postcodes (Appendix C), were randomly selected from the Australian Electronic White Pages telephone directory and contacted by telephone. The person within each household who most recently had their birthday, aged 18 years and over, was invited to participate in the study. Exclusion criteria were applied at initial phone contact. Individuals who were unable to sufficiently communicate with the telephone interviewer (e.g., language difficulties or intellectual capability), or who indicated they were too ill to participate, were excluded. The NWAHS did not recruit people residing in institutions such as nursing homes. However, individuals living in units attached to a nursing home were eligible to participate.

During the initial CATI, information regarding doctor-diagnosed health conditions including CVD, risk factors and demographics were collected from each participant, and a clinic appointment organised. Paper-based surveys were sent to participants for return at the clinic

visit or by post. These paper-based surveys collected further self-reported information including family history of cardiometabolic disease, doctor-diagnosed diabetes, smoking behaviour, and additional demographics.

Biomedical measures were collected at the clinic visits by trained clinical staff following a structured protocol. Measurements began with blood pressure as the average of two measures taken five to ten minutes apart with the participant relaxed and seated. Height (without shoes to the nearest 0.5 cm) was measured using a wall-mounted stadiometer. Weight (without shoes, light clothing, to the nearest 0.1 kg) was taken using standard digital scales. Waist and hip circumferences (nearest 0.1 cm, mean of three measures) were measured using an inelastic tape maintained in the horizontal plain. The waist measurement was taken at the narrowest part of the waist with the participant standing comfortably and weight distributed evenly between both feet. The hip circumference was measured at the maximum posterior protuberance of the buttock. A fasting blood sample (approximately 10mL) was taken to measure triglyceride, total cholesterol, high density lipoprotein, low density lipoprotein, glucose and HbA_{1c} (Grant et al. 2006).

Participants recontacted for participation in Waves 2 and 3 were similarly involved in CATIs, self-report questionnaires and clinical data collection. Further information on participant recruitment procedures has previously been published (Grant et al. 2006).

To minimise attrition rate, participants were asked to supply contact details of a close family member or friend at each wave in case they could not be contacted for future waves. Regular contact was made through an annual newsletter and birthday cards, each of which included the opportunity to update contact details. The Electronic White Pages and the South Australian and National Electoral Rolls were used to track participants and two databases on registered deaths (South Australian and National) were searched for possible matches to cohort participants (Grant et al. 2009).

Of the 8,213 individuals who were eligible to participate in the NWAHS, 5,850 completed a phone interview (CATI) and 4,056 attended a clinic assessment (49.4% of eligible sample) creating the baseline NWAHS sample (Wave 1, 2000-03; Table 5.2). Of the Wave 1 participants, 3,564 (90.1% of eligible sample) completed a Wave 2 CATI (2005-06) and 3,205 (81.0% of

eligible sample) attended the Wave 2 clinic assessment. Wave 3 (2008-10) included 2,872 CATI participants (77.4% of eligible sample) and 2,487 clinic attendees (67.0%).

Table 5.2 NWAHS sampling frame, participant numbers for each wave and reasons for sample loss

Wave	**Wave 1**	**Wave 2**	**Wave 3**
Years	**2000-03**	**2005-06**	**2008-10**
Mode	CATI/Clinic	CATI/Clinic	CATI/Clinic
Original sampling frame	10096	4056	4056
Died	-	100	292
Ineligible sample (e.g. withdrawn, too ill)	1883	NA	54
Unable to contact	NA	NA	0
Eligible Sample	8213	3957	3710
Proportion of sampling frame	*81.3%*	*97.6%*	*91.5%*
Non-contact	215	233	463
Unable to complete	-	22	0
Refused interview	2148	138	375
CATI sample	5850	3564	2872
Proportion of eligible sample	*71.2%*	*90.1%*	*77.4%*
Did not attend clinic	1794	359	385
Total participation for Wave	**4056**	**3205**	**2487**
Proportion of eligible sample	*49.4%*	*81.0%*	*67.0%*

Logistic regression was used to predict cohort attrition between waves (defined as not attending the clinic or lacking HbA_{1c} data at Waves 2 or 3). Results are shown in Table 5.3. The pattern of missingness did not meet the *missing completely at random* criterion. Participants who were male, younger, low household income, not in the work-force, currently a smoker or an ex-smoker (compared to never smoked), and not married (or de facto) were more likely to have incomplete clinic attendance and missing HbA_{1c} information at follow ups. These measures were therefore included in statistical models in Studies 2 and 3 to satisfy the analytic criterion of *missing at random* (Murray & Findlay 1988). Further details on study specific analytic samples, including sample specific cohort attrition, are provided in Section 5.5.

Table 5.3 Predictors of loss to follow-up for the complete NWAHS cohort (n=4056[#])

Variable (baseline)	Predicting incomplete clinic attendance (n=3784)		Predicting incomplete HbA_{1c} data (n=3784)	
	OR (95% CI)	P value	OR (95% CI)	P value
Sex (male)	1.23 (1.06-1.41)	0.005	1.24 (1.07-1.43)	0.003
Age	0.98 (0.98-0.99)	<0.0001	0.98 (0.98-0.99)	<0.0001
Education (not university graduate)	1.01 (0.81-1.25)	0.96	1.01 (0.82-1.27)	0.87
Household income				
Low (<$20000)	1.35 (1.05-1.74)	0.02	1.32 (1.03-1.70)	0.03
Medium ($20000-$60000)	0.99 (0.82-1.19)	0.88	0.97 (0.80-1.16)	0.71
High (>$60000)	1	-	1	-
Employment status				
Full-time employed	0.48 (0.39-0.58)	<0.0001	0.48 (0.39-0.58)	<0.0001
Part-time employed	0.65 (0.53-0.81)	<0.0001	0.65 (0.53-0.80)	<0.0001
Not in work-force	1	-	1	-
Smoking status				
Current smoker	1.74 (1.45-2.11)	<0.0001	1.72 (1.43-2.08)	<0.0001
Ex-smoker	1.24 (1.07-1.45)	0.005	1.24 (1.07-1.44)	0.005
Never smoked	1	-	1	-
Marital status (single)	1.43 (1.24-1.66)	<0.0001	1.44 (1.24-1.66)	<0.0001
HbA_{1c}	1.09 (0.99-1.19)	0.08	1.09 (0.99-1.19)	0.08

[#]analysis sample is less due to missing baseline data (particularly education/income/smoking)

Studies 2 and 3 in this research used spatial datasets linked based on georeference points representing NWAHS participants' residential addresses. Of the 4,056 Wave 1 NWAHS participants, 15 had an invalid residential address for which georeference points could not be created, resulting in a Wave 1 sample of 4041 georeferenced participants. Details of the NWAHS participants and samples used in each of the studies are provided in Section 5.5 and Chapters 6-8.

5.4.2 2007 South Australian Retail Database

Data extracted from the South Australia Retail Database (Department of Planning, Transport and Infrastructure; DPTI 2007a) were used to construct contextual measures included in Studies 2 and 3, namely, net retail area used in constructing the walkability index (Study 2), and fast food and healthful food availability (Study 3).

The South Australia Retail Database (DPTI 2007a), catalogues shops within the Adelaide Statistical Division and major centres in the Outer Adelaide Statistical Division, with information including: shop location; retail activity type; and shop gross floor space (m^2). Shop retail activity type is coded according to predominant retail activities (Spatial Planning Analysis and Research Unit 2008). Contextual food environment data were extracted according to these retail codes (Appendix D). The primary purpose of the Retail Database was for strategic planning and guidance in development of retail policy (Spatial Planning Analysis and Research Unit 2008). Data were collected over a five-month period (April to August) in 2007 and included all retailers where three or more shops were situated next to each other, or where a shop (or shops) had gross floor-space greater than 200m^2. Shops were identified from the 1999 Retail Database, investigation of relevant zones (i.e., commercial, local shopping, neighbourhood centre zones etc.), street directories, Telstra Yellow Pages® telephone directory, and consultation with local experts. Field based assessments were undertaken by a team of surveyors to compile shop data. Further information relating to the 2007 Retail Database, including a full list of the treatment of retail activities and data capture is available (see Spatial Planning Analysis and Research Unit 2008).

5.4.3 2007 South Australian Property Cadastre

Data extracted from the 2007 South Australian Property Cadastre (DPTI 2007b) were used to calculate land use mix and dwelling density measures used in constructing the walkability index (Study 2). Additionally, the validation of the 2007 Adelaide POS dataset (Section 5.4.5) used 2007 Property Cadastre data.

The 2007 South Australian Property Cadastre (DPTI 2007b) provides a state-wide record of all land parcels, including parcel boundaries, value and associated land use codes. Data are collected by licensed surveyors and the database is maintained by the South Australian Government for taxation purposes. Four-digit land use codes provide detailed information on the type and use for a given parcel (e.g., 1100 residential house, 4110 vacant allotment, 4500 reserve, 7130 cricket, 7900 recreation).

5.4.4 2006 Adelaide StreetPro™ Road Data

Intersection density, defined as the count of intersections with greater than two potential travel directions divided by buffer area in hectares, was used in constructing the walkability

index (Study 3). Intersection density was constructed from 2006 Adelaide StreetPro™ Road Data (Pitney Bowes Business Insight 2007) included as part of MapInfo Professional software (version 12.0.2, Pitney Bowes, Connecticut, USA).

5.4.5 2007 Adelaide Public Open Space Dataset

POS availability (Study 3) was calculated from data extracted from the validated 2007 Adelaide POS dataset. Constructed from the 2011 Adelaide POS dataset (Daker 2014) and the 2007 South Australian Property Cadastre (DPTI 2007b), the 2007 Adelaide POS dataset was validated using information (land use codes) from the 2007 Property Cadastre, and historical imagery (2007) from NearMap (NearMap Australia Pty Ltd), Google Earth and StreetView™ (Google Inc, USA). A description of the methods used to construct and validate the dataset are provided in Section 5.7.2.2.3.

5.4.6 South Australian Monitoring and Surveillance System

Compositional health-related norms measures (Studies 2 and 3) were constructed from data extracted from the SAMSS. The SAMSS is administered by the Population Research and Outcome Studies (PROS) Unit, The University of Adelaide, on behalf of the South Australian Government, SA Health. By monitoring population trends in chronic diseases and their risk factors, the SAMSS provides important data to support planning, implementation, and evaluation of health services and programs (PROS 2012). SAMSS data are collected across South Australia using CATIs, with approximately 7200 participants per year, selected by most recent birthday within households randomly selected from the Electronic White Pages Telephone directory (PROS 2012). Individuals of all ages are included, with information on infants and children in the household collected using an appropriate surrogate process (PROS 2012). However, SAMSS data included for use within this research were restricted to adults (18 years and over) residing within the study region of interest (i.e., urban north-west Adelaide as defined in Section 5.2). The SAMSS includes self-reported data on physical activity behaviour, fruit and vegetable intake, and height and weight.

Data used in this research were linked using a GIS and georeferences. However, geocoding (i.e., transforming location information such as residential address into a georeference point) permissions were not sought from SAMSS participants prior to 2006, thus earlier data could not be used. Data were pooled for the years 2006 to 2010 to achieve greater SAMSS

participant representation within the project spatial units (defined in Section 5.6). Overall, the response rate for SAMSS contacts during 2006-2010 was 65% with 35,830 interviews conducted across South Australia. Of the 8355 SAMSS participants 18 years and over residing within the NWAHS region (i.e., SAMSS participants whose data were eligible for use in constructing local descriptive norms), 6860 participant records were geocoded (82%; 1439 participants did not provide consent [17%] and 56 [<1%] could not be geocoded).

5.4.7 2006 Population and Housing Census

Four area-level socioeconomic measures were constructed from 2006 Australian Bureau of Statistics (ABS) Census data: 1) proportion of persons with a university education; 2) median weekly household income; 3) proportion unemployed; and 4) the Index of Relative Socio-Economic Disadvantage (IRSD) from the ABS Socio-Economic Indexes for Areas (SEIFA). Each of the four measures was considered for use in Studies 2 and 3 to represent area-level SES. Details on their operationalisation and selection for use in Studies 2 and 3 are provided in Section 5.7.2.3.1.

Australian Census data are collected every five years with the objective to accurately measure the number and key characteristics of people and dwellings in Australia on Census Night. Census data are used for a broad range of purposes by governments, businesses, academics, researchers, students, community organisations and individuals (ABS 2007). Census forms are completed by household members, with one member able to fill in the Census form on behalf of the household. Respondent error is reduced through form design, focus group testing, field testing, awareness campaigns and help available through the accompanying 'Census Guide', website and telephone help line. Recording of Census information is automatic using scanning (Intelligent Character Recognition) software, and quality assurance procedures include sample checking and corrections to minimise processing error. For more information on collection and processing of Australian Census data see Trewin (2006).

5.5 Participants: NWAHS cohort

Demographics for the NWAHS sample at baseline are shown in Table 5.4. Relative to the 2001 Adelaide metropolitan region (usual resident population), there were no statistically significant differences (Chi square) by sex, education or household income. However, older individuals were over-represented and younger individuals under-represented.

Table 5.4 Sociodemographic characteristics of geocoded NWAHS participants at baseline compared with 2001 Population Census for the Adelaide metropolitan region

Sociodemographic characteristic		NWAHS (2000-2003; n=4041)	Adelaide (ABS 2001a)	P value (Chi square)
Age	18-29 years	11.4%	20.6%	<0.01
	30-44 years	28.1%	29.1%	0.18
	45-64 years	38.0%	31.0%	<0.01
	65 years and over	22.5%	19.2%	<0.01
Sex	Male	47.8%	49.0%	0.12
Education	University graduate	11.9%	11.3%	0.23
Household income	<AUD$20000 per annum	31.5%	30.6%	0.24
	≥AUD$20000 per annum	68.5%	69.4%	0.24

NB: Percentages may not sum to 100% due to rounding; Due to differences in data collection question response categories between the 2001 Census and NWAHS, marital status data were not directly comparable and no comparison was conducted.

Each of the three studies within this dissertation included different analytic samples drawn from the NWAHS. Due to differences in study design between Study 1 and Studies 2 and 3, derivation of the Study 1 analytic sample is presented separately to that for Studies 2 and 3.

Study 1 included six sets of analytic samples drawn solely from NWAHS data. Sample loss due to exclusion criteria and missing data is shown in Table 5.5. Further information on Study 1, including a description of the sample at baseline is provided in Chapter 6.

Derivation of the three analytic samples used in Studies 2 and 3 is shown in Table 5.6 (analytic samples highlighted in bold). Further description of each of the studies, including baseline characteristics of the analytic samples is provided in Chapters 7 and 8.

Table 5.5 Derivation of Study 1 analytic samples for men and women

Sample	Men			Women		
Baseline NWAHS (n=4056)	1933			2123		
Baseline cCICR and CVD/T2DM data (n=3993)	1898			2095		
	CVD	**T2DM**	**CMD**	**CVD**	**T2DM**	**CMD**
Disease free at baseline	1699	1680	1535	1975	1925	1844
Loss due to no 10-year incident data	563	648	603	618	722	717
Loss due to incomplete FRS data	12	98	97	9	113	103
Analytic sample	**1124**	**934**	**835**	**1348**	**1091**	**1024**
Disease Incidence	130	106	153	122	96	159
Incidence rate (%)	11.57	11.35	18.32	9.05	8.80	15.53

Abbreviations: CVD – cardiovascular disease; T2DM – type 2 diabetes; CMD – cardiometabolic disease; FRS – Framingham Risk Score

Table 5.6 Derivation of analytic samples used in Studies 2 and 3 based on application of inclusion criteria

	n	Reason for reduced numbers
NWAHS Wave 1	4056	-
Geocoded (Baseline)	4041	15 participants with invalid residential addresses
Residing in urban area (Baseline)	3887	154 participants outside of urban area
Did not change residential address between Wave 1 and Wave 2	3322	565 participants moved between Wave 1 and Wave 2
State Suburbs with ≥10 participants	3173	149 participants resided in suburbs with ≤9 other participants
CVD/diabetes free at baseline	2621	552 participants reported CVD/diabetes at baseline
At least one set of HbA_{1c} and individual-level covariates data	2582	39 participants lacked at least one set of HbA_{1c} and individual-level covariates data
Contextual features	2213	369 participants lacked area POS availability or walkability data
Compositional features		
Prevalence of overweight/obesity (BMI≥25kg/m^2)	**1890**	323 participants lacked local overweight/obesity norm data
Prevalence of physical inactivity (<150 minutes/week)	**1906**	307 participants lacked local physical inactivity norm data
Prevalence of insufficient fruit intake (< 2 serves daily)	**1945**	268 participants lacked local fruit intake data

Abbreviations: BMI – body mass index; CVD – cardiovascular disease; NWAHS – North West Adelaide Health Study; POS – public open space

Predictors of NWAHS cohort attrition were assessed using logistic regression within the three analytic samples (i.e., after application of inclusion criteria as listed in Table 5.6). Results, shown in Table 5.7, are similar for each of the samples. Participants who were younger, not in the work-force, currently a smoker or an ex-smoker (compared to never smoked), and not married (or de facto) were more likely to have not attended the clinic at follow up waves. This information was therefore included in analysis models as individual-level covariate data.

Table 5.7 Predictors of loss to follow-up for the analytic samples used in Studies 2 and 3

	Local overweight/obesity norm sample n=1890		**Physical inactivity norm sample n=1906**		**Insufficient fruit intake norm sample n=1945**	
Variable (baseline)	**OR (95% CI)**	**P value**	**OR (95% CI)**	**P value**	**OR (95% CI)**	**P value**
Sex (male)	1.13 (0.90-1.40)	0.2968	1.10 (0.88-1.37)	0.3877	1.11 (0.89-1.38)	0.3466
Age	0.98 (0.98-0.99)	0.0003	0.98 (0.97-0.99)	<0.0001	0.98 (0.97-0.99)	<0.0001
Education (not university graduate)	1.13 (0.81-1.57)	0.4718	1.14 (0.82-1.58)	0.4415	1.14 (0.83-1.58)	0.4213
Household income						
Low (<$20000)	0.99 (0.68-1.46)	0.9770	1.02 (0.70-1.49)	0.9140	1.03 (0.71-1.50)	0.8787
Medium ($20000-$60000)	0.91 (0.69-1.21)	0.5106	0.94 (0.71-1.24)	0.6428	0.93 (0.71-1.23)	0.6278
High (>$60000)	1	-	1	-	1	-
Employment status						
Full-time employed	0.45 (0.33-0.60)	<0.0001	0.43 (0.32-0.58)	<0.0001	0.44 (0.33-0.59)	<0.0001
Part-time employed	0.59 (0.43-0.81)	0.0011	0.55 (0.40-0.76)	0.0002	0.56 (0.41-0.77)	0.0003
Not in work-force	1	-	1	-	1	-
Smoking status						
Current smoker	1.80 (1.35-2.40)	<0.0001	1.67 (1.25-2.22)	0.0005	1.70 (1.28-2.26)	0.0003
Ex-smoker	1.50 (1.20-1.89)	0.0006	1.48 (1.18-1.87)	0.0005	1.48 (1.18-1.89)	0.0007
Never smoked	1	-	1	-	1	-
Marital status (single)	1.74 (1.39-2.18)	<0.0001	1.70 (1.35-2.12)	<0.0001	1.74 (1.39-2.17)	<0.0001
HbA_{1c}	1.05 (0.82-1.36)	0.6892	1.10 (0.85-1.41)	0.4678	1.08 (0.84-1.38)	0.5610

NB: Loss to follow-up defined as lacking HbA_{1c} data at Wave 2 or 3

5.6 Spatial units

Studies 2 and 3 applied two different spatial units, participant-centred road network buffers (environmental exposures) and State Suburbs (to account for spatial clustering in multilevel models).

5.6.1 Participant-centred road network buffers

Area-level exposures (contextual and compositional features) were defined within participant-centred road-network buffers by radiating a set distance along the road-network in all directions. Road-network distances were set at 1000m and 1600m from each NWAHS participant's residential address. These distances represent the distance covered by walking at a comfortable pace of 5km/hour for 12 minutes and 19 minutes respectively (Bohannon 1997).

Road-network distance buffers were constructed around each geocoded NWAHS participant residential address at Wave 2 using ArcMAP (version 9.3.1, ESRI, Redlands, California). Network analyst, service area was used to create buffers with distances of 1000m and 1600m along the road network away from each participant geocoded address point. Buffers were allowed to overlap and were defined as polygons including 50m either side of the road centre line. The resultant buffers were saved as a shape file and used to spatially link with datasets enabling data extraction necessary to operationalise environmental variables. The intent was to perform analyses using environmental exposures defined within the two differently sized road-network buffers. However, unstable estimates of local descriptive norms associated with smaller counts of SAMSS survey participants within the smaller buffer precluded their use (see Section 5.7.2.2.1).

5.6.2 State Suburbs

The State Suburb, was included in analysis models for Studies 2 and 3. State Suburbs are formed by aggregating CDs to align with the most recent gazetted suburb at the time of the Census (ABS 2001). State Suburbs for 2001 were used to align with Wave 1 of the NWAHS.

5.7 Measures

5.7.1 Study 1

Measures used in Study 1 included two cCICRs, two Framingham Risk Scores, metabolic syndrome status and incident CVD and type 2 diabetes. The construction of these cardiometabolic risk indicators is described in the following sub-sections.

5.7.1.1 Continuous clinical indices of cardiometabolic risk

Two cCICRs were each constructed from five cardiometabolic risk factors: waist circumference; mean arteriole pressure (calculated as MAP = [2 x diastolic blood pressure + systolic blood pressure]/3); triglycerides; fasting blood glucose; and HDL (multiplied by −1 to account for its protective effect). These risk factors have previously been used to construct indices of cardiometabolic risk (Johnson et al. 2007; Okosun et al. 2010; Paquet et al. 2010) and represent cardiometabolic risk components used to define metabolic syndrome (ATPIII 2002; IDF 2006). Prior to cCICR construction, the five risk factor measures were transformed (log 10) to improve their distribution. Each risk factor was standardised by sex to account for sex-related differences.

The first cCICR (cCICR-Z) was constructed as the mean of the standardised risk factors. A higher score signifies greater cardiometabolic risk compared to the sample population. The second cCICR (cCICR-PCA) was constructed using principal component analysis (PCA; orthogonal rotation). PCA was performed using the five standardised risk factors, stratified by sex. The risk score, cCICR-PCA, was constructed using the weighted sum of the first two principal components using the proportion of variance explained as weights following the methods of Wijndaele and colleagues (2006). These first two components explained a total of 61.69% of the variance (41.86% and 19.83%; Eigenvalues 2.09 and 0.99) among men, and 60.14% of the variance (42.75% and 18.39%; Eigenvalues 2.14 and 0.92) among women. Similarly to the cCICR-Z a higher cCICR-PCA score signifies greater cardiometabolic risk.

5.7.1.2 Framingham Risk Scores

General CVD risk (10-year risk) and type 2 diabetes risk (8-year risk) were calculated using the relevant Framingham Risk Score algorithm (D'Agostino et al. 2008; Wilson et al. 2007). General CVD risk was calculated separately for men and women as the sum of points allocated according to levels of risk factors (tables of point allocations are provided in Appendix E). Risk

factors used in risk score calculation were: age; HDL; total cholesterol; systolic blood pressure (with and without medication); current smoker; and diabetic status. Type 2 diabetes risk was similarly calculated by summation of points allocated based on levels of risk factors (provided in Appendix E), namely: fasting glucose level; BMI; HDL; triglyceride, blood pressure (or treatment for hypertension); and parental history of diabetes mellitus.

5.7.1.3 Metabolic syndrome

Metabolic syndrome status was defined using the ATPIII definition (ATPIII 2002). Participants were defined as having the metabolic syndrome based on having three of the following five risk factors: abdominal obesity (waist circumference > 102 cm for men or > 88 cm for women); elevated triglyceride level (≥ 1.7 mmol/L); reduced HDL (< 1.03 mmol/L for men or < 1.29 mmol/L for women); elevated blood pressure (≥ 130/85 mmHg); and elevated fasting plasma glucose level (≥ 6.1 mmol/L).

5.7.1.4 Incident type 2 diabetes, cardiovascular disease and cardiometabolic disease

Participants were considered to have type 2 diabetes if they had HbA_{1c} values ≥ 6.5% (48 mmol/mol) (IEC 2009), fasting plasma glucose level ≥ 7 mmol/L (IDF 2006), or self-reported previous diagnosis by a doctor. CVD was determined from self-reported doctor diagnosis only. Incident CVD was coded for participants who were CVD-free at baseline but determined to have CVD at Wave 2 or 3. Incident type 2 diabetes was similarly coded. Incident cardiometabolic disease, was constructed by combining incident CVD and type 2 diabetes.

5.7.2 Study 2 and 3

Objectives 2 and 3 were assessed in the physical activity environment (Study 2) and food environment (Study 3). NWAHS data were used to represent the outcome (HbA_{1c}; Section 5.7.2.1) and individual-level covariate information (Section 5.7.2.3.2) in both studies. Environmental measures differed between the two studies. Study 2 included local descriptive norms for overweight/obesity and physical inactivity (Section 5.7.2.2.1), and the contextual built environment features walkability (Section 5.7.2.2.2) and availability of POS (Section 5.7.2.2.3). Local descriptive norms for overweight/obesity was similarly included in Study 3, along with local descriptive norms for insufficient fruit intake (Section 5.7.2.2.1) and the contextual measures: availability of fast-food outlets and healthful food resources (Section 5.7.2.2.4). Both studies included area-level SES as a covariate (Section 5.7.2.3.1).

5.7.2.1 Outcome variable: HbA_{1c}

Cardiometabolic risk was expressed as HbA_{1c} concentration (%), assayed at each of the three NWAHS waves from fasting blood samples collected at clinic visits. HbA_{1c}, reflecting 2-3 month time-averaged blood glucose levels, is a stable marker of glycaemic control and thus cardiometabolic risk (Bennett, Guo & Dharmage 2007). Concentrations of 6.5% or greater are considered indicative of diabetes (IEC 2009). However, lower concentrations are monotonically related to all-cause mortality and incident CVD independently of other risk factors (blood pressure, cholesterol and lipid profiles, BMI, waist-to-hip ratio, cigarette smoking, and history of CVD) (Khaw et al. 2004). Further discussion of the selection of the outcome variable used in Studies 2 and 3 is provided in Chapter 6, Results 1.

5.7.2.2 Environmental exposures

Environmental exposure information for Studies 2 and 3 was expressed within 1600m participant centred buffers (Section 5.6). Geocoded data used to construct local descriptive norms were only available for 2006-2010, therefore contextual measures were expressed at 2007 to closely match the composition measures in time.

5.7.2.2.1 Local descriptive norms

Local descriptive norms were defined as the local prevalence of: 1) overweight/obesity (BMI ≥ 25 kg/m^2); 2) physical inactivity (< 150 minutes of moderate intensity exercise per week); and 3) insufficient fruit intake (< 2 serves of fruit per day) (Brown et al. 2012; National Health and Medical Research Council 2013; WHO 2014). Local prevalence was defined as the proportion of SAMSS participants per buffer classified as not meeting the health recommendation for the physical or behavioural risk factor.

The integration of SAMSS data to construct local descriptive norms measures provides a greater sample size per buffer than would be possible using the NWAHS data, enabling greater precision when defining local area health-related norms than would otherwise be possible. In addition, the use of SAMSS data to represent local descriptive norms avoids the issue of same sample bias (spurious associations due to measurement error in both reports being correlated; Diez Roux 2007). The use of data aggregated from surveys has previously been used to represent environmental features such as social cohesion and safety (Auger et al. 2008; Chaix et al. 2008; Mujahid et al. 2007).

Concordance between SAMSS participants (adults 18 years and over) and NWAHS buffers was established using a GIS and the resultant concordance table exported. Individual-level self-report SAMSS participant data identified as pertaining to each NWAHS buffer were then extracted and aggregated for each NWAHS buffer wherein buffer-specific prevalence rates were calculated. Processing of individual-level SAMSS data was performed by the data custodians (PROS Unit, The University of Adelaide) to protect the confidentiality of SAMSS participants.

To maximise SAMSS participant representation within each NWAHS participant buffer, SAMSS data were pooled across survey years 2006 to 2010. Aggregated norms data for NWAHS buffers with fewer than 50 SAMSS participants, or less than five participants per measurement category, were not released by the data custodians in order to protect confidentiality of SAMSS participants and support the reliability of estimates. The resultant sample loss was particularly severe for the 1000m buffer size, hence this unit was not considered further. Similarly, other norms measures initially considered (insufficient vegetable intake, and fruit and vegetable intake combined) suffered severe sample loss even at the 1600m buffer (e.g., fruit and vegetable combined intake norm n=750) and were therefore dropped from further consideration.

Unstandardised prevalence rates were used following the precedent of Blok and colleagues (Blok et al. 2013). Standardisation of within buffer data based on Adelaide population distributions of age and sex would be inappropriate as these factors (age and sex) vary spatially and the aim was to represent what individuals were exposed to in their local environments. As such, standardisation of SAMSS data based on available weightings (e.g., for the Adelaide metropolitan region) may reduce the representativeness of the local area measures, artificially reducing or inflating spatial variation, and potentially result in over adjustment of models.

5.7.2.2.2 Walkability

A walkability index was constructed using data drawn from the Adelaide 2007 StreetPro™ Road Data (Pitney Bowes Business Insight 2007), the 2007 South Australian Property Cadastre (DPTI 2007b), and the 2007 South Australian Retail Database (DPTI 2007a).

The index was calculated using methods previously applied within the NWAHS region (Coffee et al. 2013), based on Leslie and colleagues (2007). For each NWAHS participant buffer, walkability was calculated as the sum of deciles for four components representing connectivity (intersection density) and proximity (dwelling density, land use mix and net retail area) (Coffee 2005; Frank et al. 2005; Leslie et al. 2007). Walkability values range from 4 to 40, lower values representing less walkable environments. Brief descriptions of the walkability components are provided below, further detail is available from Coffee and colleagues (2013).

Intersection density was defined as the count of intersections (defined as nodes with travel directions greater than two) by NWAHS buffer area (intersection count/area of buffer intersected parcels in km^2). Dwelling density was calculated as the count of dwellings by the total residential area within each NWAHS participant buffer (dwelling count/area of buffer intersected residential parcels in km^2). Land use mix was calculated as an entropy index using three types of land use: residential; commercial; and recreation;

$$Entropy = -\sum_k [(p_k)(\ln p_k)]/(\ln N)$$

where *K* is the category of land use; *P* is the proportion of the land area within the road-network buffer categorised as a specific land use; and *N* is the number of land-use categories. Based on ecological modelling, the entropy index provides a measure of diversity with a score of 0-1 where 0 indicates homogeneity (i.e., only one type of land use is present) and 1 represents heterogeneity (even distribution among land-use categories) (Coffee 2005; Frank, Engelke & Schmid 2003; Leslie et al. 2007). Net retail area, reflecting shopping centre pedestrian accessibility (Cervero & Kockelman 1997; Frank et al. 2006; Gebel, Bauman & Owen 2009), was defined as gross retail space by the total retail parcel area (gross retail area/total retail parcel area). Lower values indicate larger parking area to retail ratio and less pedestrian friendly access.

5.7.2.2.3 Public open space

POS was defined as land parcels greater than $150m^2$ available for public use, including local, linear (e.g., off-road cycling or walking path), recreational, national and conservation parks, botanic gardens, coastal areas, and outdoor sports facilities (e.g., sports fields, tennis courts).

POS availability was expressed as the proportion of buffer area (POS area/ buffer intersected land parcel area).

The 2007 Adelaide POS dataset was constructed in MapInfo Professional (version 12.0.2, Pitney Bowes, Connecticut, USA) from the validated 2011 Adelaide POS dataset (Daker 2014) and the 2007 South Australian Property Cadastre (DPTI 2007b). Methods used in construction and validation of the 2011 Adelaide POS dataset are described in detail by Daker (2014).

To construct and validate the 2007 Adelaide POS dataset, the 2007 Adelaide POS dataset was overlain on the validated 2011 Adelaide POS dataset in a GIS. Land parcels identified as POS in the 2011 POS dataset were checked against 2007 Property Cadastre land use codes and historical imagery (2007) from NearMap (NearMap Australia Pty Ltd), Google Earth and StreetView™ (Google Inc, USA) to confirm they were POS in 2007. Additional land parcel data were extracted from the 2007 Property Cadastre according to land use code (codes provided in Appendix F) to identify potential 2007 POS not already included in the validated 2011 POS dataset. Such land parcels were similarly validated using historical imagery and added (where appropriate) to the validated 2007 POS dataset.

Land parcels not meeting the above definition of POS were removed prior to finalising the validated 2007 dataset. Excluded land parcels included: divided-road median strips and road-side vegetation, car parks, sports stadiums not publicly accessible (e.g., Adelaide Oval), residential, vacant land (industrial, primary production), and water catchment. Where two or more POS parcels had adjacent borders with no intervening accessibility issue (e.g., a fence or roadway), these parcels were merged to create one larger parcel more accurately representing the physical POS. Validation of the entire dataset was performed separately by two researchers using MapInfo Professional (version 12.0.2, Pitney Bowes, Connecticut, USA). Coding differences were resolved by consensus. A third researcher was consulted when consensus was not achieved. The use of imagery such as Google Earth and StreetView™ in validation of POS has been used previously (Taylor et al. 2011).

Concordance between POS land parcels and NWAHS participant buffers was established in MapInfo Professional (version 12.0.2, Pitney Bowes, Connecticut, USA) using the ‘intersect’ function. POS data were extracted according to this concordance table. Additional data

management (e.g., calculating per buffer POS counts, POS area, and proportions) was then performed using Microsoft Access 2010 (Microsoft Corporation, Washington, USA).

5.7.2.2.4 Food resource availability

Fast food and healthful food resource availability were each expressed as counts per buffer and density measures (count/area of buffer intersected parcels in km^2).

Contextual food resource data were extracted from the 2007 South Australian Retail Database (DPTI 2007a) based on retail codes provided in Appendix D. Food resources were classified as providing fast food or healthful food based on these retail codes, using classifications designed by a dietician for use in a previous Australian study (Turrell & Giskes 2008). Fast-food outlets were defined as major fast-food franchises (e.g., McDonalds©) and independent fast-food take-away stores (e.g., fish and chips). Healthful food resources were defined as greengrocers, butchers, supermarkets (with > $200m^2$ floor space), and health food shops. Food outlets selling a mix of healthful and unhealthful foods, with neither food group being obviously predominant (e.g., sandwich and lunch bars, bakeries, and restaurants other than those identified as fast food), were excluded from classification.

Road-network distance from NWAHS participants' residence to food resources was calculated using Network Spatial Analyst in ArcGIS (version 9.3.1, ESRI, Redlands, California). Healthful food resources and fast-food outlets identified within 1600m of participant residences were then summed according to type. Density measures (count/area of buffer intersected parcels in km^2) were calculated in addition to counts.

5.7.2.3 Covariates

5.7.2.3.1 Area-level covariate: socioeconomic status

Area-level SES data were extracted from the 2006 Australian Population and Housing Census (ABS 2006c) at the smallest available unit, the CD. CDs were used during the 2006 Census of Population and Housing data collection period and include an average of 220 dwellings (ABS 2006a). To protect confidentiality, Census data are only released in aggregated form and the smallest available spatial unit for these data is the CD. Consequently, it was not possible to express these data within NWAHS participant-centred road-network buffers and an alternative approach was used.

Concordance between NWAHS participant road-network buffers and CDs was established based on dwelling points (from the 2007 Adelaide Property Cadastre) using MapInfo software (version 12.0.2, Pitney Bowes, Connecticut, USA). The resultant concordance table contained a listing of dwellings within CDs associated with each road-network buffer. This concordance table was subsequently imported into Microsoft Access 2010 (Microsoft Corporation, Washington, USA). CD-level data were further aggregated using the weighted average of values from CDs intersected by the NWAHS participant buffers. Weights were defined based on the proportion of dwellings within a CD included within the NWAHS buffer:

$$BUFFER_{SES} = \sum \left[CD_{SES} \times \frac{dwellings_a}{dwellings_b} \right]$$

where $dwellings_a$ represents the number of dwellings included within a CD intersected with a buffer, and $dwellings_b$ represents the total number of dwellings within a buffer. This process created a CD-derived value representing area-level SES for each NWAHS participant. This method assumes that the distribution of the characteristic of interest (area-level SES) is evenly distributed across all dwellings which is an improvement over assuming that the characteristic is spread evenly across the area of the spatial unit with no recognition of the distribution of dwellings.

Area-level education (proportion of persons with a university degree or higher) represented area-level SES in Studies 2 and 3. Four area-level SES measures were initially considered: 1) proportion of persons with a university degree or higher; 2) median weekly household income; 3) proportion unemployed; and 4) the SEIFA-IRSD. The SEIFA-IRSD is derived from the ABS Census (in this case for 2006) and constructed from multiple variables including area-level income, education, employment type, dwelling type and family structure (ABS 2006b). The SEIFA-IRSD is intended as a general expression of SES and the index has commonly been employed within Australian studies (e.g., Adams et al. 2009).

Though composite indicators such as the SEIFA-IRSD are commonly used to represent area-level SES, the use of composite indicators prevents inference regarding specific area-level SES effects (Ngo et al. 2014). Further, use of country-specific composite scores reduces comparability across studies in different countries (Diez Roux & Mair 2010) and composite

index construction (e.g., SEIFA scores) changes over time limiting comparability of studies using the same index at different time periods (ABS 2008).

Use of a single (i.e., not composite), clearly defined measure was therefore preferred. Area-level education was the prime candidate. Education is commonly used to indicate SES in epidemiological studies as it tends to be established early in life and does not vary as can income, employment or occupation, and preliminary models using area-level education had the best fit (compared with other area-level SES measures) based on Akaike's Information Criterion (AIC), Bayesian Information Criterion (BIC) and -2 log likelihood (see below). Each of the four area-level SES measures were moderately to highly correlated with each other as shown in Table 5.8. Therefore, only one area-level SES variable was included in Study 2 and 3 analysis models to avoid potential multicollinearity issues.

Table 5.8 Correlations between area-level SES variables for the included analytic samples

	SEIFA-IRSD	Household income (median)	Education (proportion with university degree)	Unemployment rate
Overweight/obesity norm analytic sample (n=1890)				
SEIFA-IRSD	1.00	0.86	0.65	-0.90
Household income (median)	0.86	1.00	0.38	-0.77
Education (proportion with university degree)	0.65	0.38	1.00	-0.51
Unemployment rate	-0.90	-0.77	-0.51	1.00
Physical inactivity norm analytic sample (n=1906)				
SEIFA-IRSD	1.00	0.87	0.66	-0.91
Household income (median)	0.87	1.00	0.40	-0.78
Education (proportion with university degree)	0.66	0.40	1.00	-0.52
Unemployment rate	-0.91	-0.78	-0.52	1.00
Insufficient fruit intake norm analytic sample (n=1945)				
SEIFA-IRSD	1.00	0.87	0.66	-0.91
Household income (median)	0.87	1.00	0.39	-0.77
Education (proportion with university degree)	0.66	0.39	1.00	-0.52
Unemployment rate	-0.91	-0.77	-0.52	1.00

All correlations statistically significant at p<0.0001

Model fit was assessed in a series of preliminary multilevel models (as per Study 2 and 3 analyses described in Section 5.8.2), including one of the four area-level SES measures with time and the individual-level covariates (Section 5.7.2.3.2). Models using area-level education had the best fit based on AIC, BIC and -2 log likelihood (Table 5.9) supporting the decision to select area-level education to represent area-level SES.

Table 5.9 Model fit statistics for different expressions of area-level SES in multilevel models assessing 10-year change in HbA_{1c}

Expression of area-level SES	F value	P value	-2 Log Likelihood	AIC	BIC
Overweight/obesity norm analytic sample (n=1890)					
No area-level SES variable	-	-	4284.6	4312.6	4348.9
SEIFA-IRSD score	0.01	0.9143	4284.6	4314.6	4353.5
Income (median weekly household income)	0.03	0.8622	4284.6	4314.6	4353.5
Education (proportion of persons with university education)	2.91	0.0912	4281.8	4311.8	4350.7
Unemployment rate	0.02	0.8809	4284.6	4314.6	4353.5
Physical inactivity norm analytic sample (n=1906)					
No area-level SES variable	-	-	4345.1	4373.1	4409.5
SEIFA-IRSD score	0.20	0.6551	4344.9	4374.9	4413.8
Income (median weekly household income)	0.07	0.7948	4345.1	4375.1	4414.0
Education (proportion of persons with university education)	3.45	0.0663	4341.8	4371.8	4410.7
Unemployment rate	0.17	0.6832	4345.0	4375.0	4413.9
Insufficient fruit intake norm analytic sample (n=1945)					
No area-level SES variable	-	-	4402.9	4430.9	4467.2
SEIFA-IRSD score	0.08	0.7816	4402.8	4432.8	4471.8
Income (median weekly household income)	0.00	0.9835	4402.9	4432.9	4471.8
Education (proportion of persons with university education)	3.27	0.0735	4399.7	4429.7	4468.7
Unemployment rate	0.08	0.7760	4402.8	4432.8	4471.8

NB: Models include time and individual-level covariates

5.7.2.3.2 Individual-level covariates

Individual-level covariates included in Study 2 and 3 analysis models were selected based on previous empirical research and analyses of cohort attrition between NWAHS waves as described in Section 5.5. Individual-level covariates were: age; sex; employment status (full-time, part-time, or not in the work force); level of education (university graduate or not); marital status (married/de facto, or single); and smoking status (current smoker, ex-smoker, or never smoked). Covariates other than baseline age and sex were treated as time varying.

5.8 Analytic approaches

5.8.1 Study 1

Objective 1 was divided into two studies, each assessing one component of the objective:

Study 1a) to validate a composite index of cardiometabolic risk constructed solely from clinical measures; and

Study 1b) to assess the utility of the composite index of cardiometabolic risk in estimating change in cardiometabolic risk over time

Study 1a, the first published manuscript, used odds ratios, true positive rate, false positive rate, receiver operating characteristics (ROC) curves, and the area under the curve (AUC) to assess the discriminant ability of the cCICRs and Framingham risk scores to predict 10-year incident CVD and/or type 2 diabetes.

Odds ratios, indicating the strength of association between the risk indicators and outcomes (i.e., disease incidence) were calculated for the continuous risk scores (cCICRs and Framingham Risk Scores) using sex-specific age-adjusted logistic regression models predicting each outcome (incident CVD and/or type 2 diabetes). Continuous risk scores were standardised (by sex) prior to analysis to allow for comparisons of odds ratios across regression models. For metabolic syndrome, odds ratios were calculated using two-by-two frequencies tables.

As a strong association (i.e., a large odds ratio) between outcome and predictor does not necessarily imply good predictive accuracy in correctly classifying an individual (Pepe et al. 2004), ROC curves were generated by plotting true positive rate by false positive rate across the possible range of values for each risk score. True positive rate (i.e., sensitivity) is the probability of a positive test outcome for a diseased individual while false positive rate (i.e., 1-specificity) is the probability of a positive test outcome for a non-diseased individual. A perfect risk score would have a true positive rate of 1 and false positive rate of 0 (Pepe et al. 2004). The AUCs were then calculated, providing a measure of predictive accuracy. An AUC of 0.5 represents prediction equal to chance, an AUC of 1.0, perfect prediction (Greiner, Pfeiffer & Smith 2000). The resultant AUCs were statistically compared to assess differences in the predictive accuracies of the cCICRs and the Framingham Risk Scores. For comparison purposes,

true positive rate for each of the risk scores was calculated by fixing false positive rate at the levels for metabolic syndrome (ATPIII definition; ATPIII 2002) in predicting CVD and/or type 2 diabetes. False positive rate for each score was similarly calculated. All analyses were conducted in STATA (version 12.1, StataCorp, Texas, USA).

Study 1b assessed the utility of the cCICR-Z in estimating change in cardiometabolic risk over time. Preliminary assessment of longitudinal performance of the cCICR-Z, its component risk factors, and HbA_{1c}, was done using linear multilevel models as per the analyses for Studies 2 and 3 (see the following section for details), with the sample restricted to those identified as healthy (i.e., CVD and type 2 diabetes free) at baseline. As cardiometabolic risk is accepted to increase with increasing age (D'Agostino et al. 2008; Kahn et al. 2005; Leiter et al. 2011), it was expected that the mean of the cCICR-Z, its component factors, and HbA_{1c}, would each increase over time, as the cohort aged.

Multilevel models are a flexible method to model longitudinal relationships, even in studies with unequal observations per case and unequally spaced data (repeated measures not equidistant) (West, Welch & Galecki 2007). The longitudinal nature of the data, with repeated observations over time nested within cases (participants), and the clustering of participants within geographical location, creates issues of non-independence due to correlations between measures. Multilevel models can account for this non-independence (Diez Roux 2002).

For Study 1b, models included three levels: time (level 1); nested within participants (level 2); who were themselves nested within State Suburbs (level 3). At level 1, time-specific cardiometabolic risk data (i.e., cCICR-Z, component risk factors, or HbA_{1c}) were regressed on time of measurement (in years) from baseline data collection. As data collection between participant waves was unevenly spaced, with slightly different years possible within each wave, time was expressed in a continuous format from the participant's first clinic visit. Random effects, specified at level 2, were included to allow variability in baseline participant cardiometabolic risk (intercept) and change in cardiometabolic risk over time (slope for time). Level 3 specified a random intercept to account for variations in baseline cardiometabolic risk across State Suburbs. These analyses were conducted in SAS (version 9.4, SAS Institute Inc, Cary, North Carolina).

5.8.2 Studies 2 and 3

Analyses for Studies 2 and 3 were performed using linear multilevel models. The models included three levels, with time nested within cases (participants) which were themselves nested within State Suburbs. At the first level (time level) time-specific HbA_{1c} data were regressed on time of measurement (in years) from baseline data collection. Model level two (participant level) modelled the participant baseline values of, and changes in, HbA_{1c} as a function of contextual and compositional features. Random effects were specified to allow variability in baseline HbA_{1c} concentrations (intercept) and change (slope for time) across participants as a function of individual-level predictors. Lastly, a third level accounted for spatial clustering within State Suburbs, with a random intercept (third level intercept) specified to allow for variations in baseline values in HbA_{1c} across State Suburbs.

Separate sets of models were constructed by sequentially adding variables as follows (see Table 5.10 for details of variable combinations by study and model set): 1) compositional norm, time, and the two-way interaction between these terms (compositional norm x time); 2) context, and the two-way interaction term (context x time); and 3) area-level education (covariate). Individual-level covariates (including predictors of cohort attrition) were included in all models. Two-way interaction terms for predictors and time (e.g., compositional norm x time) assessed the influence of the predictor (e.g., compositional norm) on *change* in HbA_{1c} over time. Additional two-way (compositional norm x context) and three-way (compositional norm x context x time) interaction terms were included in full models to test for interactions between environmental predictors in relation to baseline HbA_{1c} and *change* in HbA_{1c} respectively. Environmental measures were standardised prior to analyses to allow comparison of their relative effects. All analyses were conducted using SAS (version 9.4, SAS Institute Inc, Cary, North Carolina). Statistical significance was set at alpha = 0.05.

Table 5.10 Model construction and variable inclusions for Studies 2 and 3

Set	Model 1	Model 2	Model 3	Model 4
	Composition + Composition x Time	**+ Context + Context x Time**	**+ Area-level Education**	**+ Composition x Context + Composition x Context x Time**
	Study 2: Physical activity environment			
1	Overweight/obesity norm + Overweight/obesity norm x Time	+ Walkability + Walkability x Time	+ Area-level Education	+ Overweight/obesity norm x Walkability + Overweight/obesity norm x Walkability x Time
2	Overweight/obesity norm + Overweight/obesity norm x Time	+ POS availability + POS availability x Time	+ Area-level Education	+ Overweight/obesity norm x POS availability + Overweight/obesity norm x POS availability x Time
3	Physical inactivity norm + Physical inactivity norm x Time	+ Walkability + Walkability x Time	+ Area-level Education	+ Physical inactivity norm x Walkability + Physical inactivity norm x Walkability x Time
4	Physical inactivity norm + Physical inactivity norm x Time	+ POS availability + POS availability x Time	+ Area-level Education	+ Physical inactivity norm x POS availability + Physical inactivity norm x POS availability x Time
	Study 3: Food environment			
1	Overweight/obesity norm + Overweight/obesity norm x Time	+ Fast food availability + Fast food availability x Time	+ Area-level Education	+ Overweight/obesity norm x Fast food availability + Overweight/obesity norm x Fast food availability x Time
2	Overweight/obesity norm + Overweight/obesity norm x Time	+ Healthful food availability + Healthful food availability x Time	+ Area-level Education	+ Overweight/obesity norm x Healthful food availability + Overweight/obesity norm x Healthful food availability x Time
3	Insufficient fruit intake norm + Insufficient fruit intake norm x Time	+ Fast food availability + Fast food availability x Time	+ Area-level Education	+ Physical inactivity norm x Fast food availability + Physical inactivity norm x Fast food availability x Time
4	Insufficient fruit intake norm + Insufficient fruit intake norm x Time	+ Healthful food availability + Healthful food availability x Time	+ Area-level Education	+ Physical inactivity norm x Healthful food availability + Physical inactivity norm x Healthful food availability x Time

NB: all models include individual-level covariates and time

5.9 Summary

This chapter has summarised the overall research design, described the data sources used and operationalisation of measures, and detailed the analytic approaches used in the studies. The following three chapters present the results of this dissertation. Chapter 6, attending Objective 1, includes the first of the published manuscripts and follow-up study. Chapters 7 and 8 are the second and third published manuscripts, attending to Objectives 2 and 3 within the physical activity and food environments.

Chapter 6

Results 1

6 Results 1

The aim of this dissertation was to assess the independent and interactive contributions of compositional norms and contextual features to change in cardiometabolic risk. As such, an appropriate expression of cardiometabolic risk was needed. This chapter, addressing research Objective 1, outlines the selection of the outcome measure used in Studies 2 and 3.

Objective 1 was divided into two studies, each assessing one element of the objective:

Study 1a) to validate a composite index of cardiometabolic risk constructed solely from clinical measures (Section 6.1); and

Study 1b) to assess the utility of the composite index of cardiometabolic risk in estimating change in cardiometabolic risk over time (Section 6.2).

6.1 Study 1a: Validation of the continuous clinical index of cardiometabolic risk

Study 1a is the first published manuscript. A statement of authorship signed by each contributing author and copyright information is provided in Appendix G. The reference for the published paper is:

Carroll, S, Paquet, C, Howard, N, Adams, R, Taylor, A, & Daniel, M 2014, 'Validation of continuous clinical indices of cardiometabolic risk in a cohort of Australian adults', *BMC Cardiovascular Disorders*, vol. 14, no. 1, p. 27, DOI: 10.1186/1471-2261-14-27

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http://bmccardiovascdisord.biomedcentral.com/articles/10.1186/1471-2261-14-27

Carroll et al. BMC Cardiovascular Disorders 2014, 14:27
http://www.biomedcentral.com/1471-2261/14/27

BMC Cardiovascular Disorders

RESEARCH ARTICLE Open Access

Validation of continuous clinical indices of cardiometabolic risk in a cohort of Australian adults

Suzanne J Carroll[1*], Catherine Paquet[1,2], Natasha J Howard[1], Robert J Adams[3], Anne W Taylor[3] and Mark Daniel[1,4]

Abstract

Background: Indicators of cardiometabolic risk typically include non-clinical factors (e.g., smoking). While the incorporation of non-clinical factors can improve absolute risk prediction, it is impossible to study the contribution of non-clinical factors when they are both predictors and part of the outcome measure. Metabolic syndrome, incorporating only clinical measures, seems a solution yet provides no information on risk severity. The aims of this study were: 1) to construct two continuous clinical indices of cardiometabolic risk (cCICRs), and assess their accuracy in predicting 10-year incident cardiovascular disease and/or type 2 diabetes; and 2) to compare the predictive accuracies of these cCICRs with existing risk indicators that incorporate non-clinical factors (Framingham Risk Scores).

Methods: Data from a population-based biomedical cohort (n = 4056) were used to construct two cCICRs from waist circumference, mean arteriole pressure, fasting glucose, triglycerides and high density lipoprotein: 1) the mean of standardised risk factors (cCICR-Z); and 2) the weighted mean of the two first principal components from principal component analysis (cCICR-PCA). The predictive accuracies of the two cCICRs and the Framingham Risk Scores were assessed and compared using ROC curves.

Results: Both cCICRs demonstrated moderate accuracy (AUCs 0.72 – 0.76) in predicting incident cardiovascular disease and/or type 2 diabetes, among men and women. There were no significant differences between the predictive accuracies of the cCICRs and the Framingham Risk Scores.

Conclusions: cCICRs may be useful in research investigating associations between non-clinical factors and health by providing suitable alternatives to current risk indicators which include non-clinical factors.

Keywords: Cardiometabolic, Cardiovascular disease, Type 2 diabetes, Risk scores, ROC, AUC, Validation

Background

Various indicators of cardiometabolic risk are accepted for use in population health studies. Well-known examples include the Framingham Risk Scores for cardiovascular disease (CVD) and type 2 diabetes. While such indicators can have both clinical and research utility, composite indicators of risk typically include non-clinical factors such as behaviour (e.g., smoking in the Framingham 10-year General CVD Risk Score), family history (e.g., ASSIGN, Framingham Diabetes Risk Score), or area-level disadvantage (e.g., ASSIGN, QRISK) [1-4]. The inclusion of these non-clinical factors in composite outcomes usually improves absolute risk prediction. This approach however, can have limitations for inferential research.

For instance, in studies that evaluate the mechanisms by which area-level characteristics (e.g., area-level disadvantage) influence cardiometabolic risk, non-clinical factors are often framed as mediators and thus must be excluded from composite expressions of cardiometabolic risk that are evaluated as outcomes [5]. A variable cannot be both a predictor and an outcome. Adapting established indicators of cardiometabolic risk by removing non-clinical components can reduce the utility of such risk indicators and necessitate re-validation. Composite measures incorporating only clinical factors to

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Indicators of cardiometabolic risk typically include non-clinical factors (e.g., smoking). While the incorporation of non-clinical factors can improve absolute risk prediction, it is impossible to study the contribution of non-clinical factors when they are both predictors and part of the outcome measure. Metabolic syndrome, incorporating only clinical measures, seems a solution yet provides no information on risk severity. The aims of this study were: 1) to construct two continuous clinical indices of cardiometabolic risk (cCICRs), and assess their accuracy in predicting 10-year incident cardiovascular disease and/or type 2 diabetes; and 2) to compare the predictive accuracies of these cCICRs with existing risk indicators that incorporate non-clinical factors (Framingham Risk Scores).

Methods

Data from a population-based biomedical cohort (n = 4056) were used to construct two cCICRs from waist circumference, mean arteriole pressure, fasting glucose, triglycerides and high density lipoprotein: 1) the mean of standardised risk factors (cCICR-Z); and 2) the weighted mean of the two first principal components from principal component analysis (cCICR-PCA). The predictive accuracies of the two cCICRs and the Framingham Risk Scores were assessed and compared using ROC curves.

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Both cCICRs demonstrated moderate accuracy (AUCs 0.72 – 0.76) in predicting incident cardiovascular disease and/or type 2 diabetes, among men and women. There were no significant differences between the predictive accuracies of the cCICRs and the Framingham Risk Scores.

Conclusion

cCICRs may be useful in research investigating associations between non-clinical factors and health by providing suitable alternatives to current risk indicators which include non-clinical factors.

Keywords

Cardiometabolic, Cardiovascular disease, Type 2 diabetes, Risk scores, ROC, AUC, Validation

6.1.1 Background

Various indicators of cardiometabolic risk are accepted for use in population health studies. Well-known examples include the Framingham Risk Scores for cardiovascular disease (CVD) and type 2 diabetes. While such indicators can have both clinical and research utility, composite indicators of risk typically include non-clinical factors such as behaviour (e.g., smoking in the Framingham 10-year General CVD Risk Score), family history (e.g., ASSIGN, Framingham Diabetes Risk Score), or area-level disadvantage (e.g., ASSIGN, QRISK) [1-4]. The inclusion of these non-clinical factors in composite outcomes usually improves absolute risk prediction. This approach however, can have limitations for inferential research.

For instance, in studies that evaluate the mechanisms by which area-level characteristics (e.g., area-level disadvantage) influence cardiometabolic risk, non-clinical factors are often framed as mediators and thus must be excluded from composite expressions of cardiometabolic risk that are evaluated as outcomes [5]. A variable cannot be both a predictor and an outcome. Adapting established indicators of cardiometabolic risk by removing non-clinical components can reduce the utility of such risk indicators and necessitate re-validation. Composite measures incorporating only clinical factors to express cardiometabolic risk are required to support the investigation of associations between cardiometabolic risk and sociodemographic and behavioural factors.

One composite indicator of cardiometabolic risk based solely on clinical risk factors is metabolic syndrome. Current definitions of metabolic syndrome incorporate counts of risk factors exceeding established threshold values wherein individuals are classified as either having, or not having the syndrome [6,7]. However, the underlying expression of each risk measure is continuous, and cardiometabolic risk a progressive function of these combined risk measures [8-10]. Using current methods for defining metabolic syndrome, for example the International Diabetes Federation (IDF) and Adult Treatment Panel III (ATPIII) definitions [6,7], a minimal change in risk measures can result in a change of classification status [10], yet a large change may not. A continuous index of risk constructed from clinical factors would eliminate this issue and more closely approximate an individual's actual continuum of risk, providing information relating to risk severity. Evidence supports the use of a continuous clinical index of cardiometabolic risk (cCICR) in health studies [11,12] and the use of such a

measure has been recommended by the American Diabetes Association and the European Association for the Study of Diabetes [8].

Different methods of constructing a cCICR, typically from metabolic syndrome components, have been employed. These cCICRs include: a count of risk factors exceeding recommended thresholds [13]; the sum of risk factor points established by risk factor deciles [14]; the sum or mean of z-scores [11,12,15]; and components derived by principal component analysis [16,17]. While incorporating a count of risk factors improves the utility of metabolic syndrome status as an expression of risk, this strategy still does not account for the progressive nature of risk within each risk factor. Using deciles to express each risk factor is a progressive approach but still compromises statistical power and distributional information due to categorisation [18]. Arguably, the most progressive and efficient cCICRs are those involving the summation of z-scores and principal component analysis derived scores. A cCICR constructed as the mean of standardised metabolic syndrome components has been validated for use in children and adolescents [11,15], though not in adult populations. However, an alternative method utilising principal component analysis has been validated for use in adult populations [16,17]. No study thus far has constructed such cCICRs for use in adult populations and evaluated their validity. The aims of this study were: 1) to construct two cCICRs and assess their accuracy in predicting 10-year incident CVD and/or type 2 diabetes in a longitudinal adult cohort; and 2) to compare these cCICRs with existing risk indicators comprising clinical, sociodemographic and behavioural factors (Framingham CVD [F-CVD] and Diabetes [F-T2DM] Scores).

6.1.2 Methods

This research study was part of the Place and Metabolic Syndrome (PAMS) Project which aims to assess the mechanisms that explain the relationships between place, health behaviour and cardiometabolic health. The PAMS Project received ethical approval from the University of South Australia, Central Northern Adelaide Health Service, Queen Elizabeth Hospital, and South Australian Department for Health and Aging Human Research Ethics Committees.

6.1.2.1 Participants

The North West Adelaide Health study (NWAHS), a 10-year biomedical cohort comprised of 4056 randomly selected men and women aged 18 years and over, provided data for this study. The NWAHS has involved three waves of data collection thus far, Wave 1 (2000–03, n = 4056), Wave 2 (2005–06, n = 3563), and Wave 3 (2008–10, n = 2871). Each wave has involved the collection of standardised measures using Computer-Assisted Telephone Interviews, self-report paper questionnaires, and clinic visits. Written informed consent was obtained from all participants prior to each wave of data collection. Further information on recruitment has previously been published [19].

6.1.2.2 Measures

Biomedical data

Biomedical data were collected during the hospital-based clinic visits at each wave. Measures included blood pressure (mean of two measures), height and weight, waist and hip circumference (mean of three measures), and a fasting blood sample which was used to determine triglyceride, total cholesterol, high density lipoprotein (HDL), glucose and glycosylated haemoglobin (HbA_{1c}) concentrations. Participants were asked about previously diagnosed type 2 diabetes, heart attack, stroke, angina, or transient ischaemic attack/mini stroke. Further details of data collection are available elsewhere [19].

Risk scores

Risk factor measures used to construct cCICRs were transformed (log 10) to improve their distribution. The five factors used in the construction of the cCICRs were: waist circumference, mean arteriole pressure (MAP = [2DBP + SBP]/3), triglycerides, fasting blood glucose, and HDL (multiplied by −1 to account for its protective effect). These risk factors have previously been used to construct cCICRs [13,15] and represent cardiometabolic risk components used to

define metabolic syndrome [6,7]. Each risk factor was sex-standardised to account for sex-related differences.

Mean of z-scores (cCICR-Z)

A cCICR was constructed as the mean of the standardised risk factors. A higher score signifies greater cardiometabolic risk compared to the sample population.

Principal component analysis (cCICR-PCA)

A second cCICR was constructed using principal component analysis (orthogonal rotation). Principal component analysis was performed using the five standardised risk factors, stratified by sex. The risk score, cCICR-PCA, was constructed using the weighted sum of the first two principal components with the proportion of variance explained as weights following the methods of Wijndaele and colleagues [16]. These first two components explained a total of 61.69% of the variance (41.86% and 19.83%; Eigenvalues 2.09 and 0.99) among men, and 60.14% of the variance (42.75% and 18.39%; Eigenvalues 2.14 and 0.92) among women.

Framingham Risk Scores

General CVD risk (10-year risk) and type 2 diabetes risk (8-year risk) were calculated using the relevant Framingham Risk Score algorithm [1,3].

Incident type 2 diabetes and cardiovascular disease

Participants were considered to have type 2 diabetes if they had HbA_{1c} values ≥ 6.5% (48 mmol/mol) [20], fasting plasma glucose level ≥ 7 mmol/L, or self-reported previous diagnosis by a doctor. CVD was determined from self-reported doctor diagnosis only. Incident CVD was coded for participants who were CVD-free at baseline but determined to have CVD at Wave 2 or 3. Incident type 2 diabetes was similarly coded. A third outcome measure, cardiometabolic disease, was constructed by combining incident CVD and type 2 diabetes.

6.1.2.3 Analyses

The ability of the risk scores to predict 10-year incident CVD and/or type 2 diabetes was assessed using odds ratios, true positive rate, false positive rate, receiver operating characteristics (ROC) curves, and the area under the curve (AUC). True positive rate (i.e., sensitivity) is the probability of a positive test outcome for a diseased individual while false positive rate (i.e., 1-specificity) is the probability of a positive test outcome for a non-diseased

individual. A perfect risk score would have a true positive rate of 1 and false positive rate of 0 [21].

Odds ratios were calculated to provide a measure of the strength of association between indicators of risk and incident CVD and/or type 2 diabetes. For metabolic syndrome, odds ratios were calculated using two-by-two frequencies tables. For the continuous risk scores (cCICRs and Framingham Risk Scores), odds ratios were calculated using sex-specific age-adjusted logistic regression models predicting each outcome. Continuous risk scores were standardised (by sex) prior to analysis to allow for comparisons of odds ratios across regression models.

As a strong association (e.g., a large odds ratio) between outcome and predictor does not necessarily imply good predictive accuracy in correctly classifying an individual [21], ROC curves were therefore generated by plotting true positive rate by false positive rate across the possible range of values for each risk score. The AUCs were then calculated, providing a measure of predictive accuracy. An AUC of 0.5 represents prediction equal to chance, an AUC of 1.0, perfect prediction [22].

Differences between the predictive accuracies of the cCICRs and the Framingham Risk Scores were assessed by statistically comparing the AUCs. For comparison purposes, true positive rate for each of the risk scores was calculated by fixing false positive rate at the levels for metabolic syndrome (ATPIII definition [7]) in predicting CVD and/or type 2 diabetes. False positive rate for each score was similarly calculated. All analyses were conducted in STATA (version 12.1, StataCorp, Texas, USA).

6.1.3 Results

At baseline, 1898 men and 2095 women (total n = 3993) had complete clinical measures and information on CVD and type 2 diabetes status (Table 6.1). Men demonstrated worse profiles than women in regard to weight, waist girth, proportion overweight, glucose, triglycerides, HDL, blood pressure measures, and the proportion identified as having metabolic syndrome, type 2 diabetes, and CVD. Women demonstrated a worse profile than men in regard to proportion obese.

Sample loss and sample size used for models predicting the different outcomes are shown in Table 6.2. The AUCs, true positive rates, false positive rates, and odds ratios for each of the risk scores in predicting 10-year incident disease are shown in Table 6.3. As metabolic syndrome status is categorical, direct comparison of the odds ratios for metabolic syndrome status and the continuous risk scores in predicting cardiometabolic outcomes is not possible.

Table 6.1 Baseline characteristics of the sample according to sex

Characteristic	Men (n=1898)	Women (n=2095)	P value	Total (n=3993)
Age (years)	50.80 (16.76)	50.20 (16.05)	0.25	50.49 (16.39)
Weight (kg)	85.32 (15.98)	72.25 (15.74)	<0.0001	78.46 (17.38)
Waist girth (cm)	98.43 (13.03)	87.09 (14.12)	<0.0001	92.47 (14.74)
BMI (kg/m^2)	27.89 (4.78)	27.75 (6.04)	0.41	27.82 (5.48)
Overweight (BMI 25.00 – 29.99 kg/m^2) [1]	874 (46.05%)	663 (31.65%)	<0.0001	1537 (38.49%)
Obese (BMI ≥ 30 kg/m^2) [1]	512 (26.98%)	632 (30.17%)	0.03	1144 (28.65%)
Glucose (mmol/L) [2]	5.1 (4.8-5.6)	4.9 (4.5-5.3)	<0.0001	5.00 (4.6-5.5)
Triglycerides (mmol/L) [2]	1.3 (0.9-2.0)	1.1 (0.8-1.6)	<0.0001	1.20 (0.9-1.8)
HDL cholesterol (mmol/L) [2]	1.2 (1.0-1.4)	1.5 (1.2-1.7)	<0.0001	1.30 (1.1-1.6)
Total cholesterol (mmol/L) [2]	5.1 (4.4-5.8)	5.2 (4.6-6.0)	0.0001	5.2 (4.5-5.9)
Systolic blood pressure (mmHg)	130.90 (17.61)	125.50 (19.15)	<0.0001	128.03 (18.63)
Diastolic blood pressure (mmHg)	82.65 (9.96)	78.79 (10.04)	<0.0001	80.63 (10.19)
Mean arteriole pressure (mmHg)	98.72 (11.20)	94.35 (11.89)	<0.0001	96.43 (11.77)
Metabolic syndrome (ATPIII) [1]	553 (29.14%)	461 (22.00%)	<0.0001	1014 (25.39%)
Type 2 diabetes [1]	218 (11.49%)	170 (8.11%)	0.0003	388 (9.72%)
Cardiovascular disease [1]	199 (10.48%)	120 (5.73%)	<0.0001	319 (7.99%)
Cardiometabolic disease [1]	363 (19.13%)	251 (11.98%)	<0.0001	614 (15.38%)

Mean (SD) t-test; [1] proportions and Chi square; [2] median (Q1-Q3) and Wilcoxon Signed Rank
Abbreviations: BMI – body mass index; ATPIII – Adult Treatment Panel III definition

Table 6.2 Sample loss and sample used in models predicting each outcome among men and women

	Men			Women		
	CVD	**T2DM**	**CM disease**	**CVD**	**T2DM**	**CM disease**
Disease free at baseline	1699	1680	1535	1975	1925	1844
Loss due to no 10-year incident data	563	648	603	618	722	717
Loss due to incomplete FRS data	12	98	97	9	113	103
Total sample	1124	934	835	1348	1091	1024
Incidence	130	106	153	122	96	159
Incidence rate (%)	11.57	11.35	18.32	9.05	8.80	15.53

Abbreviations: CVD – cardiovascular disease; T2DM – type 2 diabetes; CM – cardiometabolic; FRS – Framingham Risk Score

Table 6.3 Associations and predictive accuracies (95% CIs) for indicators of risk predicting disease incidence (age-adjusted models)

	CVD				T2DM				CM disease			
	AUC	**TPR [1]**	**FPR [2]**	**OR**	**AUC**	**TPR [1]**	**FPR [2]**	**OR**	**AUC**	**TPR [1]**	**FPR [2]**	**OR**
Men	**n=1124**				**n=934**				**n=835**			
MetS [3]	-	0.41 (0.32-0.50)	0.27 (0.24-0.29)	1.90 (1.31-2.78)	-	0.52 (0.42-0.61)	0.22 (0.20-0.25)	3.72 (2.46-5.63)	-	0.42 (0.34-0.50)	0.21 (0.18-0.24)	2.69 (1.86-3.89)
cCICR-Z	0.73 (0.69-0.77)	0.54 (0.44-0.64)	0.16 (0.08-0.24)	1.62 (1.33-1.99)	0.76 (0.71-0.81)	0.59 (0.49-0.70)	0.17 (0.11-0.23)	2.48 (1.90-3.25)	0.75 (0.71-0.79)	0.50 (0.40-0.59)	0.15 (0.10-0.20)	2.03 (1.62-2.55)
cCICR-PCA	0.73 (0.69-0.78)	0.59 (0.49-0.69)	0.15 (0.09-0.22)	1.65 (1.35-2.03)	0.75 (0.71-0.80)	0.60 (0.48-0.73)	0.20 (0.16-0.25)	2.32 (1.79-3.00)	0.75 (0.71-0.79)	0.54 (0.44-0.63)	0.16 (0.12-0.20)	2.01 (1.61-2.51)
F-CVD	0.73 (0.69-0.77)	0.55 (0.46-0.65)	0.16 (0.10-0.22)	2.45 (1.63-3.68)	-	-	-	-	0.72 (0.68-0.77)	0.49 (0.40-0.58)	0.16 (0.11-0.22)	2.71 (1.78-4.13)
F-T2DM	-	-	-	-	0.79 (0.74-0.83)	0.68 (0.57-0.79)	0.15 (0.09-0.20)	2.27 (1.84-2.80)	0.75 (0.71-0.79)	0.54 (0.45-0.64)	0.15 (0.08-0.21)	1.81 (1.51-2.16)
Women	**n=1348**				**n=1091**				**n=1024**			
MetS [3]	-	0.27 (0.19-0.36)	0.19 (0.17-0.22)	1.54 (1.01-2.35)	-	0.40 (0.30-0.49)	0.13 (0.11-0.16)	4.21 (2.69-6.58)	-	0.28 (0.21-0.34)	0.13 (0.11-0.15)	2.68 (1.80-3.99)
cCICR-Z	0.76 (0.71-0.80)	0.57 (0.47-0.66)	0.06 (0.03-0.09)	1.24 (1.03-1.51)	0.73 (0.68-0.79)	0.40 (0.28-0.52)	0.14 (0.09-0.19)	2.04 (1.62-2.58)	0.74 (0.69-0.78)	0.43 (0.35-0.52)	0.06 (0.03-0.09)	1.66 (1.37-2.02)
cCICR-PCA	0.76 (0.72-0.80)	0.58 (0.49-0.68)	0.06 (0.02-0.09)	1.28 (1.05-1.56)	0.73 (0.67-0.78)	0.35 (0.24-0.47)	0.14 (0.09-0.19)	1.97 (1.56-2.48)	0.73 (0.69-0.78)	0.43 (0.34-0.52)	0.06 (0.04-0.09)	1.63 (1.34-1.97)
F-CVD	0.76 (0.72-0.80)	0.56 (0.46-0.65)	0.08 (0.05-0.10)	1.59 (1.07-2.35)	-	-	-	-	0.72 (0.67-0.76)	0.42 (0.32-0.51)	0.08 (0.05-0.11)	1.84 (1.25-2.69)
F-T2DM	-	-	-	-	0.75 (0.70-0.81)	0.46 (0.35-0.56)	0.09 (0.05-0.14)	2.00 (1.65-2.42)	0.74 (0.70-0.78)	0.41 (0.32-0.50)	0.07 (0.04-0.11)	1.63 (1.38-1.93)

[1] TPR with FPR fixed at MetS levels; [2] FPR with TPR fixed at MetS levels; [3] not age-adjusted

Abbreviations: CVD – cardiovascular disease; T2DM – type 2 diabetes; CM – cardiometabolic; AUC – area under the curve; TPR – true positive rate; FPR – false positive rate; OR – odds ratio; MetS – metabolic syndrome; cCICR-Z – continuous clinical index of cardiometabolic risk constructed as the mean of standardised risk scores; cCICR-PCA – continuous clinical index of cardiometabolic risk constructed using principal component analysis; F-CVD – Framingham 10-year General CVD Risk Score; FT2DM – Framingham 8-year Diabetes Risk Score

ROC curves are shown in Figure 6.1. Both cCICRs demonstrated significant associations (odds ratios) with incident CVD among men and women, and AUCs for both models reflect moderate predictive accuracy. In both men and women, the F-CVD demonstrated moderate predictive accuracy and was not significantly different to the cCICRs in this regard. These similarities in predictive accuracies were reflected in the true positive rate and false positive rate for the different scores with similar values for true positive rates at the fixed false positive rate of 0.27 (the rate for metabolic syndrome), and similar false positive rates with true positive rate fixed at 0.41.

For type 2 diabetes, the cCICRs were significantly associated with disease incidence in both sexes, and demonstrated moderate predictive accuracies. Higher odds ratios were evident between the cCICRs and incident type 2 diabetes than for incident CVD. The F-T2DM also demonstrated moderate predictive accuracy. The true positive rate for F-T2DM was slightly higher, and false positive rate slightly lower than the rates for the cCICRs. This was reflected in the slightly higher AUCs in predicting incident type 2 diabetes among both men and women. However, the difference in AUCs between models was not significant.

Both cCICRs were significantly associated with incident cardiometabolic disease among men and women, again demonstrating moderate predictive accuracies. Similarly, the F-CVD and F-T2DM demonstrated moderate predictive accuracies. There were no statistically significant differences in predictive accuracies of the risk scores in predicting incident cardiometabolic disease among men or women. The true positive rates and false positive rates were again similar for each of the risk scores for both sexes.

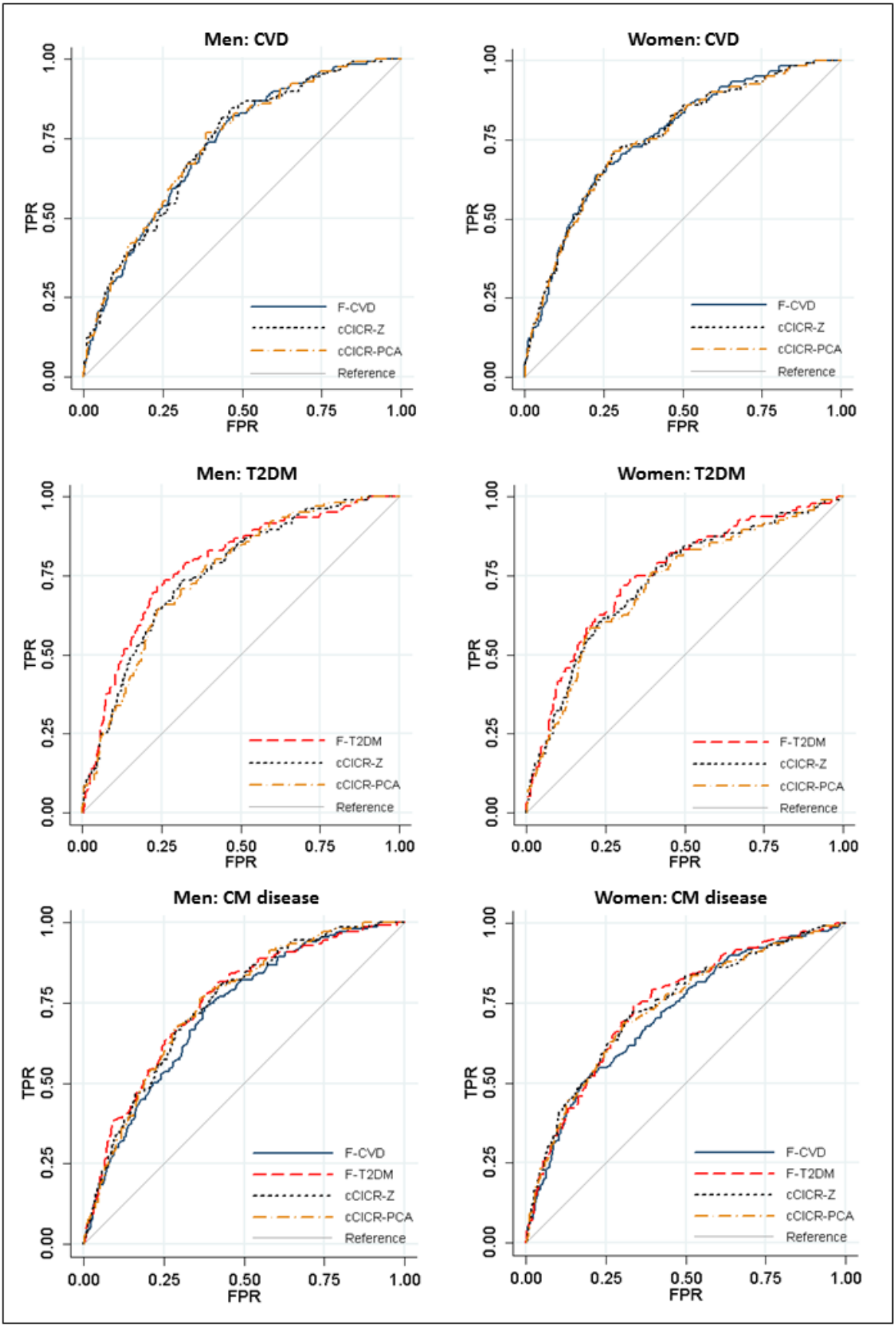

Figure 6.1 ROC curves for models predicting CVD, type 2 diabetes (T2DM) and cardiometabolic (CM) disease among men and women

6.1.4 Discussion

The objective of this study was to validate two cCICRs, developed to address limitations of dichotomous risk measures such as metabolic syndrome, and continuous indicators of cardiometabolic risk that include non-clinical factors such as behaviour. This study constructed two cCICRs that had previously been validated in either children or adult populations, and assessed their accuracies in predicting 10-year incident CVD and/or type 2 diabetes. The predictive accuracies of the cCICRs were then compared with established risk scores that incorporate sociodemographic and behavioural factors.

The two cCICRs constructed for use in this study demonstrated statistically significant associations with 10-year incident CVD, type 2 diabetes and cardiometabolic disease among men and women. Although the odds ratios for men were higher than for women for all outcomes, post-hoc analyses revealed that these differences were not statistically significantly different (p-values > 0.05).

Previous studies have assessed associations between a cCICR constructed as the first principal component of principal component analysis and incident CVD and type 2 diabetes. In one study among men and women (44–84 years) the first principal component demonstrated a hazard ratio of 1.65 (1.49-1.82) in models predicting 5.5-year CVD events, adjusted for age, sex and ethnicity [10]. Another study using the first principal component in sex-specific age-adjusted models demonstrated odds ratios of 3.4 (2.6-4.4) and 5.1 (3.6-7.2) for men and women (30–65 years) respectively in predicting 9-year incident type 2 diabetes – stronger associations than those found in the current study [17]. Associations were weaker in predicting 9-year incident CVD, with estimated odds ratios being 1.7 (1.4-2.1) and 1.7 (1.0-2.7) for men and women respectively, similar to the levels of association found in the current study. This pattern of stronger associations when predicting type 2 diabetes to weaker associations with CVD is not unexpected. Studies have found clinical indicators of risk, including metabolic syndrome status and a count of metabolic syndrome risk components, to have stronger associations with incident type 2 diabetes than incident CVD [23,24].

The differences in the strengths of associations in predicting incident type 2 diabetes between this study and that of Hillier and colleagues [17] may be due to differences in the age range of study participants, the current study including individuals as young as 18 years. Neither

previous study reported information on model fit or predictive accuracy. No study has yet examined the association and accuracy of a cCICR constructed as a mean of standardised risk factors in predicting incident CVD and/or type 2 diabetes in adults.

Both cCICRs demonstrated moderate accuracy in predicting incident CVD, type 2 diabetes and cardiometabolic disease, performing similarly well to the Framingham Risk Scores. These findings are interesting as the F-CVD and F-T2DM both include other well established risk factors in their construction. However, differences in the strength of association are noted for the F-CVD in predicting CVD and may be attributed to the inclusion of smoking status in the risk algorithm.

The two cCICRs constructed in this study performed similarly well to the Framingham Risk Scores and thus constitute viable expressions of cardiometabolic risk. Furthermore, these cCICRs can be used to represent a combined CVD and type 2 diabetes risk (i.e., cardiometabolic risk) though associations are stronger for type 2 diabetes alone. As such, cCICRs may be useful in research studies that require a cardiometabolic risk measure that is constructed solely from clinical measures.

This study found little difference in the performance of the two differently constructed cCICRs. Therefore, the choice of which measure to use within a study may depend on the design and intent of the study. The cCICR-Z places equal weight on each of the risk factors included in its construction though each of these factors may have a greater or lesser association with cardiometabolic risk. However, this does not appear to have negatively impacted the index's performance when compared to cCICR-PCA which weights the components based on a principal component analysis variance matrix. This may, however, make the cCICR-Z the more useful measure for tracking change in risk over time. Unlike the cCICR-PCA, the weightings of the components that are used to construct the cCICR-Z will not change each time the score is constructed, for example, at each time point in a longitudinal study.

A number of limitations of this study should be considered. This study made use of self-reported CVD status; type 2 diabetes status was determined based on combined self-reported status and clinical measures. However, any effect this may have had on the strengths of associations and predictive accuracies of the indicators in predicting incident CVD and type 2 diabetes will apply equally for each of the risk indicators.

The analyses in this study featured age-adjusted models. Other potential risk factors may also affect the associations reported, notably smoking status. The exclusion of models adjusting for such behavioural (and other) factors is intentional as the purpose of this study was to assess the predictive accuracy of indicators of cardiometabolic risk constructed solely from clinical risk factors. Studies intending to use cCICRs can adjust for additional factors as deemed appropriate.

In addition to the above, other clinical risk factors have demonstrated associations with cardiometabolic risk including proinflammatory and prothrombotic markers [8]. The inclusion of such risk factors may improve the accuracies of cCICRs in predicting incident cardiometabolic disease. However, these measures were not available for the current study.

Medication status (being on antihypertensive or lipid-lowering medications) was not considered in the construction and analyses of the cCICRs. Only a small number of participants were taking these medications, providing only small sample sizes for analyses of predictive accuracy. For example, models predicting cardiometabolic disease incidence amongst men taking medications (antihypertensive or lipid-lowering medications) would have included a sample of only 94 individuals. As such analyses stratified by medication status were not performed. However, post-hoc sensitivity analyses excluding individuals on antihypertensive or lipid-lowering medications suggested no notable difference in the AUCs for any of the models (results not reported). This is consistent with results from two studies validating a cCICR constructed using principal component analysis among adults [10,17] which both concluded that patterns of association were similar when performing sensitivity analyses excluding participants taking antihypertensive or lipid-lowering medications.

The cCICRs constructed in this study are sample specific, providing a relative measure of risk, each individual's risk being compared to the population sample being assessed. The Framingham Risk Scores provide estimates of absolute risk and would be the preferred choice of measures wherever absolute CVD or type 2 diabetes risk is the objective. The relative nature of the cCICRs may limit comparison across studies in different populations. This may also have implications for their use in longitudinal studies where the sample may change over time due to loss to follow-up.

6.1.5 Conclusions

This study constructed two cCICRs and assessed their associations with, and accuracy in prediction of 10-year incident CVD and/or type 2 diabetes. The predictive accuracies of these indices were compared with Framingham Risk Scores which include clinical, sociodemographic and behavioural factors in their risk algorithms. The cCICRs demonstrated similar abilities to the Framingham Risk Scores in predicting incident CVD, type 2 diabetes, and cardiometabolic disease. Little difference was noted in the performance of the two cCICRs constructed using different methods, however the cCICR-Z may be easier to use and interpret. Either cCICR may be useful in research investigating associations between cardiometabolic risk and sociodemographic or behavioural factors. Both cCICRs provide a suitable alternative to current indicators of risk which either include non-clinical factors or lack information on risk severity.

Abbreviations

AUC, Area under the curve; cCICR, Continuous clinical index of cardiometabolic risk; cCICR-PCA, A cCICR constructed using principal component analysis; cCICR-Z, A cCICR constructed as the mean of standardised risk scores; CVD, Cardiovascular disease; DBP, Diastolic blood pressure; F-CVD, Framingham 10-year General CVD Risk Score; F-T2DM, Framingham 8-year Diabetes Risk Score; FPR, False positive rate; HbA_{1c}, Glycosylated haemoglobin; HDL, High density lipoprotein; MAP, Mean arteriole pressure; MetS, Metabolic syndrome; NWAHS, North West Adelaide Health Study; OR, Odds ratio; PAMS Project, Place and Metabolic Syndrome Project; ROC, Receiver operating characteristics; SBP, Systolic blood pressure; TPR, True positive rate

Competing interests

The authors declare that they have no competing interest.

Authors' contributions

SJC, MD, CP, NJH, AWT, and RJA conceived and designed the study. SJC analysed the data under guidance of CP. SJC, CP, MD contributed to interpretation of results. SJC wrote the manuscript, and CP, NJH, and MD revised it critically for important intellectual content. All authors approved the final manuscript.

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References

1. D'Agostino RB, Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, Kannel WB: **General cardiovascular risk profile for use in primary care.** *Circulation* 2008, **117**(6)**:**743–753.

2. Hippisley-Cox J, Coupland C, Vinogradova Y, Robson J, May M, Brindle P: **Derivation and validation of QRISK, a new cardiovascular disease risk score for the United Kingdom: prospective open cohort study.** *BMJ* 2007, **335**(7611)**:**136–148.

3. Wilson PWF, Meigs JB, Sullivan L, Fox CS, Nathan DM, D'Agostino RB Sr: **Prediction of incident diabetes mellitus in middle-aged adults: the Framingham offspring study.** *Arch Intern Med* 2007, **167**(10)**:**1068–1074.

4. Woodward M, Brindle P, Tunstall-Pedoe H: **Adding social deprivation and family history to cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended Cohort (SHHEC).** *Heart* 2007, **93:**173–176.

5. Daniel M, Moore S, Kestens Y: **Framing the biosocial pathways underlying associations between place and cardiometabolic disease.** *Health Place* 2008, **14**(2)**:**117–132.

6. IDF: **IDF Diabetes Atlas**. 5th edition. Brussels: International Diabetes Federation; 2011.

7. Adult Treatment Panel III: **Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report.** *Circulation* 2002, **106**(25)**:**3143–3421.

8. Kahn R, Buse J, Ferrannini E, Stern M: **The metabolic syndrome: time for a critical appraisal.** *Diabetes Care* 2005, **28**(9)**:**2289–2304.

9. Klein BEK, Klein R, Lee KE: **Components of the metabolic syndrome and risk of cardiovascular disease and diabetes in Beaver Dam.** *Diabetes Care* 2002, **25**(10)**:**1790–1794.

10. Agarwal S, Jacobs DR Jr, Vaidya D, Sibley CT, Jorgensen NW, Rotter JI, Chen YDI, Liu Y, Andrews JS, Kritchevsky S, Goodpaster B, Kanaya A, Newman AB, Simonsick EM, Herrington, DM: **Metabolic syndrome derived from principal component analysis and incident cardiovascular events: The multi ethnic study of atherosclerosis (MESA) and health, aging, and body composition (Health ABC).** *Cardiol Res Pract* 2012, **1:** art. no. 919425. doi:10.1155/2012/919425.

11. Eisenmann JC, Laurson KR, DuBose KD, Smith BK, Donnelly JE: **Construct validity of a continuous metabolic syndrome score in children.** *Diabetol Metab Syndr* 2010, **2**(1)**:**8–16.

12. Franks PW, Ekelund U, Brage S, Wong M-Y, Wareham NJ: **Does the association of habitual physical activity with the metabolic syndrome differ by level of cardiorespiratory fitness?** *Diabetes Care* 2004, **27**(5)**:**1187–1193.

13. Paquet C, Dube L, Gauvin L, Kestens Y, Daniel M: **Sense of mastery and metabolic risk: moderating role of the local fast-food environment.** *Psychosoc Med* 2010, **72:**324–331.

14. Aguilar-Salinas CA, Rojas R, Gonzalez-Villalpando C, Gómez-Pérez FJ, Mehta R, Olaiz G, Rull JA, Cox DR: **Design and validation of a population-based definition of the metabolic syndrome.** *Diabetes Care* 2006, **29**(11)**:**2420–2426.

15. Okosun IS, Lyn R, Davis-Smith M, Eriksen M, Seale P: **Validity of a continuous metabolic risk score as an index for modeling metabolic syndrome in adolescents.** *Ann Epidemiol* 2010, **20**(11)**:**843–851.

16. Wijndaele K, Gaston B, Duvigneaud N, Matton L, Duquet W, Thomis M, Lefevre J, Philippaerts RM: **A continuous metabolic syndrome risk score [abstract].** *Diabetes Care* 2006, **29**(10)**:**2329.

17. Hillier TA, Rousseau A, Lange C, Lepinay P, Cailleau M, Novak M, Calliez E, Ducimetiere P, Balkau B: **Practical way to assess metabolic syndrome using a continuous score obtained from principal components analysis.** *Diabetologia* 2006, **49**(7)**:**1528–1535.

18. Ragland DR: **Dichotomizing continuous outcome variables: dependence of the magnitude of association and statistical power on the cutpoint.** *Epidemiology* 1992, **3**(5)**:**434–440.

19. Grant J, Chittleborough C, Taylor A, Dal Grande E, Wilson D, Phillips P, Adams R, Cheek J, Price K, Gill T, Ruffin R: **The North West Adelaide Health Study: detailed methods and baseline segmentation of a cohort for chronic diseases.** *Epidemiol Perspect Innov* 2006, **3**(4). art. no 4. doi:10.1186/1742-5573-3-4.

20. IEC: **International Expert Committee Report on the role of the A1C assay in the diagnosis of diabetes.** *Diabetes Care* 2009, **32**(7)**:**1327–1334.

21. Pepe MS, Janes H, Longton G, Leisenring W, Newcomb P: **Limitations of the odds ratio in gauging the performance of a diagnostic, prognostic, or screening marker.** *Am J Epidemiol* 2004, **159**(9)**:**882–890.

22. Greiner M, Pfeiffer D, Smith RD: **Principles and practical application of the receiver-operating characteristic analysis for diagnostic tests.** *Prev Vet Med* 2000, **45**(1–2)**:**23–41.

23. Stern MP, Williams K, González-Villalpando C, Hunt KJ, Haffner SM: **Does the metabolic syndrome improve identification of individuals at risk of type 2 diabetes and/or cardiovascular disease?** *Diabetes Care* 2004, **27**(11)**:**2676–2681.

24. Wannamethee SG, Shaper AG, Lennon L, Morris RW: **Metabolic syndrome vs Framingham risk score for prediction of coronary heart disease, stroke, and type 2 diabetes mellitus.** *Arch Intern Med* 2005, **165**(22)**:**2644–2650.

6.2 Study 1b: Assessment of the utility of the cCICR in estimating change in cardiometabolic risk over time

6.2.1 Background

The first section of this chapter (Section 6.1; Carroll, SJ et al. 2014) described the construction of two cCICRs, assessing their associations with, and accuracy in prediction of, 10-year incident CVD and/or type 2 diabetes within the NWAHS cohort. The predictive accuracies of the cCICRs were compared with those of the Framingham CVD and Diabetes Risk Scores (D'Agostino et al. 2008; Wilson et al. 2007). Logistic regression was used to calculate odds ratios predicting disease status by Wave 3 within a sample that was CVD and diabetes free at baseline. For each of the risk indicators, predictive accuracies were assessed using ROC curves, and the AUCs compared to assess differences in their predictive accuracies. The cCICRs were both statistically significantly associated with cardiometabolic disease, and demonstrated moderate accuracy in predicting cardiometabolic risk. The cCICRs performed similarly to the Framingham Risk Scores.

Though the cCICRs performed similarly well to the Framingham Scores, their ability to assess change in cardiometabolic risk over time was not evaluated. The validation was based on "caseness" over time, that is, baseline cardiometabolic risk was used to predict "having" or "not having" the disease by Wave 3 using simple logistic regression. These analyses did not explicitly model time, nor assess change in the cardiometabolic risk as expressed by the cCICR from baseline to Wave 2 or 3. Studies 2 and 3 assess the influence of environmental features on change in cardiometabolic risk over 10 years. It was therefore necessary to assess the appropriateness of using the cCICR to reflect change in cardiometabolic risk during this time period.

The use of a composite risk measure has been recommended over the use of single measures (Jackson et al. 2005; Perk et al. 2012). However, one particular concern is that the risk factors included within the cCICR may respond differentially to environmental exposures (Paquet et al. 2014). The use of a composite indicator to assess change over time would essentially sum these differential changes, providing information on how the individuals' global cardiometabolic risk changes. However, different mechanisms may link environmental features to risk components (Paquet et al. 2014). The use of a composite indicator would

preclude the ability to investigate these specific exposure-outcome relationships and their possible mechanisms.

An alternative would be to use a single cardiometabolic risk factor such as HbA_{1c}. HbA_{1c}, reflecting an individual's 2-3 month average of plasma glucose levels, is associated with both type 2 diabetes and CVD (Bennett, Guo & Dharmage 2007; Eeg-Olofsson et al. 2010; Khaw et al. 2004; Nathan et al. 2013; Nathan, Turgeon & Regan 2007; Selvin et al. 2010). Though cut points have been established for HbA_{1c} in relation to diabetes status, the relationship between HbA_{1c} and CVD may be linear. As such it may provide an alternative option to using a cCICR in representing change in cardiometabolic risk over time.

This chapter section reports on the assessment of the utility of the cCICR, constructed as a sum of z-scores, in estimating change in cardiometabolic risk. The performance of the cCICR over time was assessed relative to that of its' component risk factors and HbA_{1c}. Associations between cardiometabolic risk and age are well established (D'Agostino et al. 2008; Kahn et al. 2005; Leiter et al. 2011). It was therefore expected that the mean of the cCICR, its component factors, and HbA_{1c}, would each increase over time, as the cohort aged.

6.2.2 Methods

The cCICR was constructed for each of the three NWAHS cohort waves using data for participants identified as not having CVD or type 2 diabetes at baseline. At Wave 1, the cCICR was constructed as per Carroll and colleagues (2014) with the exception that the five sex-standardised z-scores for the risk factors were summed, not averaged. To calculate the cCICR at Waves 2 and 3, each of the five component measures were standardised (by sex) to the cohort's baseline (Wave 1) mean and standard deviation. Where a participant had missing data for one or more cCICR components at a particular wave, cCICR was coded as missing for that wave. A higher cCICR score represents greater cardiometabolic risk compared to the cohort sample at baseline.

Preliminary assessment of longitudinal performance was done using linear multilevel models as per the analyses intended for Studies 2 and 3, with the sample restricted to those identified as healthy (i.e., CVD and type 2 diabetes free) at baseline. The objective of these models was to assess whether time, expressed continuously from the participant's first clinic visit (in years), predicted change in cardiometabolic risk estimated using the cCICR.

Models included three levels: time (level 1); nested within participants (level 2); who were themselves nested within State Suburbs (level 3). State Suburbs were formed by aggregating CDs to align with the most recent gazetted suburb at the time of the Census (ABS 2001). Time was the only predictor included in these models. The first level of the model (time), regressed time-specific cCICR data on time of measurement (in years) from baseline data collection. The second level (participant level) modelled baseline values of, and changes in cCICR. Included random effects allowed for variability in baseline cCICR (intercept) and change (slope for time) between participants. The third level was included to account for spatial clustering within State Suburbs. A random intercept (third level intercept) was specified to allow variation in baseline values of cCICR between suburbs. For comparison purposes, similar models were run with each of the five cCICR components and HbA_{1c} as the outcome measure. All analyses were conducted using SAS (version 9.4, SAS Institute Inc, Cary, North Carolina). Statistical significance was set at alpha = 0.05.

6.2.3 Results

Simple descriptive statistics for the cCICR and its components at each wave are presented in Table 6.4. Though the mean cCICR increased (worsened) over the 10-year period (paired t-test: mean change -0.13, p=0.01; n=2122), cCICR decreased (improved) between Wave 1 and Wave 2 (n=2665, mean change -0.12, p=0.002), and increased (worsened) between Wave 2 and 3 (n=1979, mean change 0.18, p<0.0001).

Table 6.4 Descriptive statistics for the cCICR, its components, and HbA_{1c} for the NWAHS cohort at each Wave

	Wave 1	**Wave 2**	**Wave 3**
	Mean (SD)	**Mean (SD)**	**Mean (SD)**
	Median (IQR)	**Median (IQR)**	**Median (IQR)**
	n	**n**	**N**
cCICR	0.00 (3.24)	-0.01 (3.22)	0.16 (3.13)
Paired t-test:	-0.07 (-2.35-2.22)	-0.06 (-2.34-2.19)	0.08 (-2.05-2.29)
*W1-W2**; W2-W3***; W1-W3**	n=3379	n=2670	n=2123
MAP (mmHg)	95.46 (11.46)	96.16 (11.21)	94.56 (10.83)
Wilcoxen Sign Test:	94.33 (87.33-102.08)	95.00 (88.67-103.33)	94.17 (87.17-101.33)
*W1-W2^{ns}; W2-W3***; W1-W3****	n=3388	n=2682	n=2153
Triglycerides (mmol/L)	1.40 (0.91)	1.50 (1.19)	1.48 (0.92)
Wilcoxen Sign Test:	1.2 (0.8-1.7)	1.2 (0.8-1.8)	1.2 (0.9-1.8)
*W1-W2^{ns}; W2-W3***; W1-W3****	n=3385	n=2691	n=2132
Glucose (mmol/L)	4.96 (0.58)	4.94 (0.61)	5.10 (0.72)
Wilcoxen Sign Test:	4.9 (4.6-5.3)	4.9 (4.6-5.2)	5.0 (4.7-5.4)
*W1-W2^{ns}; W2-W3***; W1-W3****	n=3382	n=2689	n=2125
Waist circumference (cm)	90.91 (14.22)	93.21 (14.31)	94.21 (14.77)
Wilcoxen Sign Test:	90.3 (80.5-100.0)	93.0 (82.8-102.4)	93.6 (83.7-103.9)
*W1-W2***; W2-W3***; W1-W3****	n=3387	n=2681	n=2153
HDL (mmol/L)	1.39 (0.38)	1.47 (0.37)	1.52 (0.41)
Wilcoxen Sign Test:	1.3 (1.1-1.6)	1.4 (1.2-1.7)	1.5 (1.2-1.8)
*W1-W2***; W2-W3***; W1-W3****	n=3385	n=2691	n=2132
HbA_{1c} (%)	5.38 (0.46)	5.52 (0.48)	5.68 (0.52)
Paired t-test:	5.4 (5.1-5.7)	5.5 (5.2-5.8)	5.6 (5.4-5.9)
*W1-W2***; W2-W3***; W1-W3****	n=3381	n=2688	n=2129

NB: participants not identified as having CVD/type 2 diabetes at baseline; * p<0.05; **p<0.01; ***p<0.001; ^{ns} not statistically significantly different

Abbreviations: cCICR – continuous clinical index of cardiometabolic risk; CVD – cardiovascular disease; HbA_{1c} – glycosylated haemoglobin; HDL – high density lipoprotein; IQR – interquartile range; MAP – mean arteriole pressure; NWAHS – North West Adelaide Health Study; SD – standard deviation; W1 – Wave 1; W2 – Wave 2; W3 – Wave 3

Inspection of the cCICR components showed differential changes over time between measures (Table 6.4). Three of the five cCICR components changed between Wave 1 and Wave 3 in the expected directions (i.e., worsened), namely, blood triglyceride and glucose concentrations, and waist girth each increased, though triglyceride and glucose concentrations were not statistically significantly different between Waves 1 and 2. Contrary to expectations, the remaining two cCICR component risk factors improved between baseline and Wave 3: MAP decreased (improved) though there was no statistically significant change between Wave 1 and Wave 2; and HDL concentration increased (improved) between each wave. HbA_{1c} increased between each wave as expected.

Results of the preliminary multilevel models are shown in Table 6.5. Due to the shapes of the distributions (i.e., not normally distributed), results for cCICR component risk factors are also reported for log 10 transformed versions of the measures. Time (in years) predicted change in cCICR, that is, cCICR increased over time. However, though this association was statistically significant, the p-value (p=0.03) was quite large for such a large sample especially when considering the p-values obtained for change in the component risk factors and HbA_{1c} (p<0.0001). Though the component risk factors were each statistically significantly predicted by time (i.e., changed over time), these associations were not always in the expected directions as per the descriptive statistics reported above. For example, MAP decreased over time and HDL increased over time, suggesting that these measures improved as cohort participants aged. HbA_{1c} increased over time, that is, changed in the expected direction.

Table 6.5 Time (in years) predicting cardiometabolic risk as cCICR, component risk factors, and HbA_{1c}

	Time (years) Predicting outcome		Time (years) Predicting log 10 of outcome	
Outcome	**Beta (95% CI)**	**P-value**	**Beta (95% CI)**	**P-value**
cCICR	0.013 (0.002-0.025)	0.03	-	-
MAP (mmHg)	-0.114 (-0.163- -0.066)	<0.0001	-0.0005 (-0.0007- -0.0003)	<0.0001
Triglycerides (mmol/L)	0.011 (0.006-0.016)	<0.0001	0.0038 (0.0028-0.0048)	<0.0001
Glucose (mmol/L)	0.013 (0.010-0.017)	<0.0001	0.0011 (0.0008-0.0014)	<0.0001
HDL (mmol/L)	0.015 (0.014-0.017)	<0.0001	0.0047 (0.0043-0.0051)	<0.0001
Waist circumference (cm)	0.408 (0.368-0.448)	<0.0001	0.0019 (0.0017-0.0021)	<0.0001
HbA_{1c} (%)	0.037 (0.034-0.039)	<0.0001	-	-

NB: NWAHS cohort, healthy at baseline
Abbreviations: cCICR – continuous clinical index of cardiometabolic risk; CI – confidence interval; HbA_{1c} – glycosylated haemoglobin; HDL – high density lipoprotein; MAP – mean arteriole pressure; NWAHS – North West Adelaide Health Study

6.2.4 Discussion

The cCICR did not perform as expected over time. Though time predicted change in cCICR in multilevel models, the p-value was notably larger than that for the indicator's component risk factors and HbA_{1c}. The mean of the cCICR increased (worsened) between Waves 1 and 3 as expected, however, between Wave 1 and Wave 2, the mean decreased slightly (i.e., cardiometabolic risk improved). Three of the cCICR component factors, waist circumference, and blood triglyceride and glucose concentrations, worsened over time while two factors, MAP and HDL, improved over time. HbA_{1c} behaved as expected, that is, increased (worsened) between each wave, and change in HbA_{1c} was predicted by time (in years) in the expected direction.

These results support the findings of previous studies suggesting that cardiometabolic risk factors respond differentially to environmental and behavioural exposures (Boulé et al. 2001; Dhingra et al. 2007; Paquet et al. 2014). It is conceivable that MAP and HDL improved over time due to health behaviour change or prescription medication that did not affect (or affect so strongly) other risk factors. There is a need to better understand why these risk factors responded differently over time. If risk factors respond differentially to environmental exposures and health behaviours, assessment of associations between environmental exposures and the separate risk factors would provide greater insights into potential causal pathways than the use of a composite measure. Consequently, further research into the differential responses of risk factors to exposures is needed to understand mechanisms of influence and support intervention design tailored for specific health conditions. Such research is outside the scope of this dissertation but an important area for future research. The unexpected pattern of change over time for the cCICR and its risk components suggest that the cCICR may not be an appropriate choice of measure to represent change in cardiometabolic risk within longitudinal research. Moreover, though the cCICR was validated with its predictive accuracy evaluated, no cross-validation was performed within a separate cohort. Further investigation into the suitability of the cCICR is needed prior to use as an outcome measure in longitudinal research. Thus the use of a single risk factor was determined to be a better option for achieving research Objectives 2 and 3 of this PhD dissertation.

Though the cCICR did not behave as anticipated, HbA_{1c} increased over time as expected. HbA_{1c} is monotonically associated with all-cause mortality and CVD independently of other risk

factors, with CVD risk beginning well below the cut point for determining type 2 diabetes (Eeg-Olofsson et al. 2010; Khaw et al. 2004; Nathan et al. 2013). The strength and shape of associations between HbA_{1c} and diabetes and CVD make it a suitable alternative to the cCICR in representing change in cardiometabolic risk over time.

Research evaluating associations between environmental features and cardiometabolic outcomes has used HbA_{1c} as an outcome measure. Studies have assessed prediabetes and/or diabetes, defined using HbA_{1c}, in relation to availability and characteristics of POS (Bodicoat et al. 2014; Paquet et al. 2014), walkability and the food environment (Relative Food Environment Index) (Paquet et al. 2014), and fast food outlet (count) (Bodicoat et al. 2015). However, as HbA_{1c} is linearly associated with CVD (Eeg-Olofsson et al. 2010; Khaw et al. 2004; Nathan et al. 2013) it could be used as a continuous outcome variable. The use of a continuous outcome variable, as opposed to a categorised variable, would provide greater sensitivity and power in analyses (Ragland 1992). Few studies have assessed associations between environmental features and HbA_{1c} expressed in continuous format, though one cross-sectional US study reported HbA_{1c} increased monotonically across quartiles of neighbourhood deprivation in models adjusted for individual-level factors including income and education (Laraia et al. 2012).

6.3 Chapter conclusion

This chapter includes two studies, the first of which reported the validation of two cCICRs and the comparison of their accuracies in predicting incident cardiometabolic disease in relation to the Framingham Risk Scores for CVD and type 2 diabetes. However, this first study did not model "time" within prediction models, modelling "caseness" after 10 years with simple logistic regression and ROC curves. Further, no cross-validation within a separate dataset was performed. The second study assessed the ability of the cCICR to discriminate change in cardiometabolic risk over time, comparing its behaviour to that of its components and HbA_{1c}.

The cCICRs demonstrated adequate predictive capacity for type 2 diabetes and CVD. However, the cCICR (constructed as a sum of risk factor z-scores) did not behave as expected over time, creating doubt as to the appropriateness of using the cCICR as an outcome in longitudinal studies. Components within the cCICR changed differentially over time. This supports the premise that clinical risk factors respond differentially to environmental and behavioural exposures.

HbA_{1c} behaved as expected over the time period and is associated with both type 2 diabetes and CVD. It was therefore selected as an appropriate alternative to represent change in cardiometabolic risk within Studies 2 and 3. Though HbA_{1c} has previously been used as an outcome variable in research evaluating relationship between environmental exposures and health outcomes, it has typically been used to categorise diabetes and pre-diabetes status. Few studies have used HbA_{1c} expressed in a continuous format, though it is linearly related to CVD. The following two chapters, attending to Objectives 2 and 3 of this dissertation, use HbA_{1c} as the outcome.

Chapter 7

Results 2

7 Results 2

This chapter presents the second published manuscript which attends to Objectives 2 and 3 within the physical activity environment. Author contributions and copyright permissions information is provided in Appendix H. The reference for the published article is:

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Local descriptive norms for overweight/obesity and physical inactivity, features of the built environment, and 10-year change in glycosylated haemoglobin in an Australian population-based biomedical cohort

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ABSTRACT

Descriptive norms vary between places. Spatial variation in health-related descriptive norms may predict individual-level health outcomes. Such relationships have rarely been investigated. This study assessed 10-year change in glycosylated haemoglobin (HbA_{1c}) in relation to local descriptive norms for overweight/obesity (n = 1890) and physical inactivity (n = 1906) in models accounting for features of the built environment.

HbA_{1c} was measured three times over 10 years for a population-based biomedical cohort of adults in Adelaide, South Australia. Environmental exposures were expressed for cohort participants using 1600 m road-network buffers centred on participants' residential address. Local descriptive norms (prevalence of overweight/obesity [body mass index ≥25 kg/m^2] and of physical inactivity [<150 min/week]) were aggregated from responses to a separate geocoded population survey. Built environment measures were public open space (POS) availability (proportion of buffer area) and walkability. Separate sets of multilevel models analysed different predictors of 10-year change in HbA_{1c}. Each model featured one local descriptive norm and one built environment variable with area-level education and individual-level covariates (age, sex, employment status, education, marital status, and smoking status). Interactions between local descriptive norms and built environment measures were assessed.

HbA_{1c} increased over time. POS availability and local descriptive norms for overweight/obesity and physical inactivity were each associated with greater rates of HbA_{1c} increase. Greater walkability was associated with a reduced rate of HbA_{1c} increase, and reduced the influence of the overweight/obesity norm on the rate of increase in HbA_{1c}.

Local descriptive health-related norms and features of the built environment predict 10-year change in HbA_{1c}. The impact of local descriptive norms can vary according to built environment features. Little researched thus far, local descriptive norms may play an important role in the evolution of HbA_{1c} and thus cardiometabolic risk, over time. Further empirical research on local descriptive norms is necessary to understand how residential environments shape chronic disease risk.

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1. Background

Despite a growing awareness of relationships between features of built and social environments, individual-level risk factors and important cardiometabolic outcomes, public health interventions often focus on individual-level risk factors (Richard et al., 2011). To

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Abstract

Descriptive norms vary between places. Spatial variation in health-related descriptive norms may predict individual-level health outcomes. Such relationships have rarely been investigated. This study assessed 10-year change in glycosylated haemoglobin (HbA_{1c}) in relation to local descriptive norms for overweight/obesity (n=1890) and physical inactivity (n=1906) in models accounting for features of the built environment.

HbA_{1c} was measured three times over 10 years for a population-based biomedical cohort of adults in Adelaide, South Australia. Environmental exposures were expressed for cohort participants using 1600m road-network buffers centred on participants' residential address. Local descriptive norms (prevalence of overweight/obesity [body mass index ≥ 25kg/m^2] and of physical inactivity [< 150 minutes/week]) were aggregated from responses to a separate geocoded population survey. Built environment measures were public open space (POS) availability (proportion of buffer area) and walkability. Separate sets of multilevel models analysed different predictors of 10-year change in HbA_{1c}. Each model featured one local descriptive norm and one built environment variable with area-level education and individual-level covariates (age, sex, employment status, education, marital status, and smoking status). Interactions between local descriptive norms and built environment measures were assessed.

HbA_{1c} increased over time. POS availability and local descriptive norms for overweight/obesity and physical inactivity were each associated with greater rates of HbA_{1c} increase. Greater walkability was associated with a reduced rate of HbA_{1c} increase, and reduced the influence of the overweight/obesity norm on the rate of increase in HbA_{1c}.

Local descriptive health-related norms and features of the built environment predict 10-year change in HbA_{1c}. The impact of local descriptive norms can vary according to built environment features. Little researched thus far, local descriptive norms may play an important role in the evolution of HbA_{1c} and thus cardiometabolic risk, over time. Further empirical research on local descriptive norms is necessary to understand how residential environments shape chronic disease risk.

Keywords

Australia; cardiometabolic risk; built environment; descriptive norms; overweight; physical activity; public open space; walkability.

Highlights

- Local descriptive norms have rarely been examined in relation to health
- Local descriptive norms for overweight/obesity and physical inactivity predicted increasing HbA_{1c}
- Greater POS availability and poorer walkability predicted increasing HbA_{1c}
- The influence of local descriptive norms varied according to the built environment
- More research is needed to understand how local descriptive norms contribute to population health

7.1 Background

Despite a growing awareness of relationships between features of built and social environments, individual-level risk factors and important cardiometabolic outcomes, public health interventions often focus on individual-level risk factors (Richard et al., 2011). To some degree this reflects the challenge of changing built and social environments to be more supportive of healthful lifestyles and behaviour. It may also reflect a lesser acceptance of the credibility of public health research on place and health relationships, when the vast majority of this work is cross-sectional and with key pathways explicated only in part (Daniel et al., 2008).

Features of local areas are understood to function as environmental risk conditions, analogues of individual-level risk factors to which they contribute in exacerbating disease vulnerability for exposed individuals (Daniel et al., 2011). For example, a lack of public open space (POS) may inhibit opportunities for active recreation and thus physical activity resulting in elevated cardiometabolic risk. Both the specific features of areas (contextual features) and the aggregated characteristics of persons residing within these areas (compositional features) are associated with cardiometabolic risk (Daniel et al., 2011; Leal & Chaix, 2011). A review by Leal and Chaix (2011) concluded there were associations between aspects of the contextual built environment and obesity, the most commonly investigated cardiometabolic risk factor. For example, reasonably consistent associations were found between the absence of POS and greater obesity, and between walkability (a measure of urban form incorporating connectivity and proximity) and lesser obesity (Leal & Chaix, 2011). However, some studies reported null associations, or associations in unexpected directions. For example, one U.S. study found women with fewer recreational resources in their local-area were less likely to be obese (Boehmer et al., 2007).

Relationships between contextual features and cardiometabolic risk have been assessed in numerous studies. Of compositional features, area-level socioeconomic status (SES) has been well studied, with area-level SES now established as an important influence on individual health outcomes (Leal & Chaix, 2011). Very few studies have gone beyond framing social "composition" as anything other than area-level SES, yet there are many options for aggregating other conceptually different, spatially variable measures such as local descriptive norms. Given that descriptive health-related norms vary spatially (Blok et al., 2013) such

measures have the potential to serve as a useful operational form of population composition, beyond aggregated SES, in health and place research. Despite considerable rhetoric about the role of social norms in shaping the risk behaviours of exposed individuals (e.g., Christakis & Fowler, 2013; Rivis & Sheeran, 2003), local descriptive norms have rarely been subjected to empirical investigation in relation to disease risks or outcomes.

Cialdini's Focus Theory of Normative Conduct (Cialdini et al., 1990) distinguishes two different types of social norms, *injunctive* and *descriptive*. Injunctive norms refer to shared rules of conduct, what most other people approve or disapprove of. They specify what *ought* to be done, enjoining action through the promise of social sanctions (Cialdini et al., 1990). In contrast, descriptive norms are what most people actually do, what is typical or *normal* (Cialdini et al., 1990). A descriptive norm can be considered as the prevalence of a behaviour. In psychology research, both injunctive and descriptive norms have been established as important predictors of behaviour including physical activity. In the Theory of Planned Behaviour (Ajzen, 1991), both subjective norms (defined as what an individual believes others expect of them, i.e., the injunctive norm in Cialdini's description) and descriptive norms are believed to influence individual attitudes and behaviour (Rivis & Sheeran, 2003). Furthermore, Threshold Models of Collective Behaviour suggest that the proportion of people engaged in a behaviour within a social system, that is, the descriptive norm, influences whether an individual will engage in said behaviour (Granovetter, 1978). Finally, studies based on Social Contagion Theory have demonstrated that traits (including obesity) and behaviours (including smoking) cluster, and can "spread" from person to person within social networks (Christakis & Fowler, 2013).

Descriptive norms can be further divided into two types, *subjective* and *local* (Kormos et al., 2015). *Subjective* descriptive norms refer to the normative influence of people who are important to an individual, such as close friends and family. Thus, a subjective descriptive norm can be considered the prevalence of a behaviour within an individual's social network. Social Contagion Theory focuses on subjective descriptive norms. Conversely, *local* descriptive norms refer to the normative influence that derives from people sharing the same spatial setting regardless of any emotional connections (or lack thereof) (Kormos et al., 2015). Local descriptive norms can therefore be conceived as behavioural prevalence within a defined spatial setting, such as an individual's local residential area. Local descriptive norms are

associated with environmental behaviours such as littering and recycling (Cialdini et al., 1990; Nigbur et al., 2010). While the aforementioned Social Contagion Theory studies suggest the importance of social closeness in transmission of norms in relation to health traits and behaviours, local descriptive norms, norms defined by residential area or spatial proximity, have rarely been studied in this regard.

Blok and colleagues (2013), reported that normal weight Dutch adults were more likely to become overweight/obese when residing in areas with a greater local descriptive norm for overweight/obesity (expressed as prevalence) during 13 years of follow up, after accounting for individual factors and neighbourhood SES. The authors are aware of no other such studies. Spatial variations in descriptive health-related norms may *locally* predispose individuals towards (or against) health-related behaviours. Thus they may be of considerable relevance to understanding and informing preventative actions to reduce rising rates of cardiometabolic disease. Empirical studies are needed to assess the potential influence of local descriptive norms on individual health outcomes.

While local descriptive health-related norms may function as predisposing factors for healthful or unhealthful behaviours, features of the built environment can function as barriers or enablers. As such built environment features may influence associations between local descriptive health-related norms and health outcomes that are a function of behaviour, such as cardiometabolic risk. For example, associations between a local descriptive norm for physical activity and cardiometabolic health may be more pronounced where the built environment is supportive of physical activity, such as greater POS availability and walkability, as opposed to a built environment that is less supportive. Only a small number of studies have assessed whether associations between environmental features and cardiometabolic risk are modified by other environmental features (Leal & Chaix, 2011). No studies have reported on whether any given influence of local descriptive norms on cardiometabolic risk is modified by contextual built environment features.

The aims of this study were to assess: 1) the relative contributions of local descriptive norms for overweight/obesity and physical inactivity, and contextual measures of the built environment (POS availability and walkability) to 10-year change in HbA_{1c} (a marker of

cardiometabolic risk); and 2) whether associations between local descriptive health-related norms and change in HbA_{1c} varied according to these built environment features.

7.2 Methods

This study was part of the Place and Metabolic Syndrome (PAMS) Project, Adelaide, South Australia. The PAMS Project aimed to evaluate the role of environmental features in relation to the evolution of cardiometabolic risk. It received ethical approval from the University of South Australia, Central Northern Adelaide Health Service (Queen Elizabeth Hospital), and the South Australian Department for Health and Ageing Human Research Ethics Committees.

7.2.1 Study area

The baseline study area was the northern and western regions of metropolitan Adelaide (Figure 7.1), the capital city of South Australia, accounting for 38% of Adelaide's 1.1 million population in 2001 (ABS, 2001, 2003). These regions are of particular interest due to elevated cardiometabolic risk relative to other areas (Dal Grande et al., 2004; South Australian Department of Health, 2005).

Associations between environments, health behaviours and outcomes may differ between urban and rural regions (Wilcox et al., 2000). The study region was therefore limited to urban areas, defined as Census Collection Districts (CDs) with a population density of >200 persons per hectare (ABS, 2001).

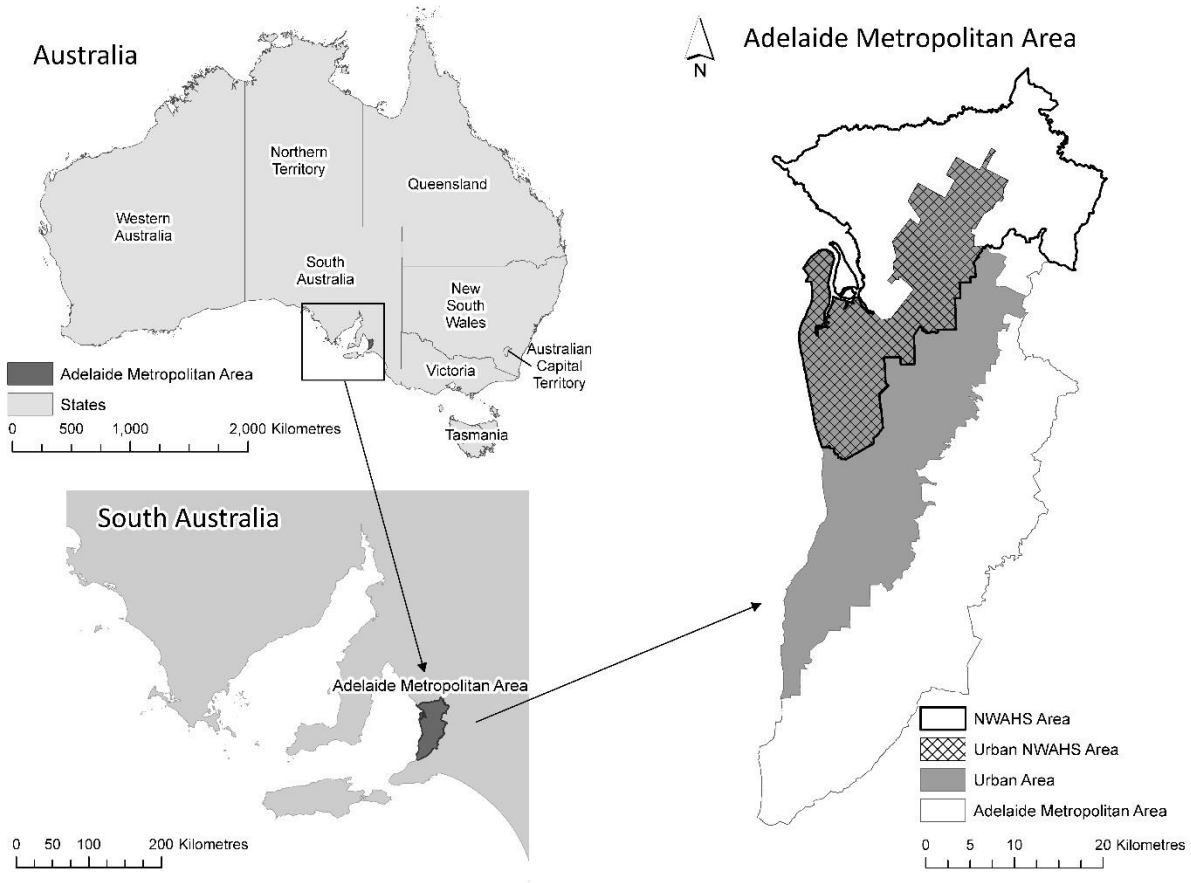

Figure 7.1 Study area – urban north-west region of Adelaide

7.2.2 Participants

The North West Adelaide Health Study (NWAHS), a 10-year biomedical cohort comprised at baseline of 4056 randomly selected men and women aged 18 years and over, provided individual-level data. Data were collected in three waves, Wave 1 (2000-03, n=4056), Wave 2 (2005-06, n=3563), and Wave 3 (2008-10, n=2871). Standardised measures, including sociodemographic and behavioural information, and residential address (used to create a geo-reference point), were collected at each wave using Computer-Assisted Telephone Interviews, self-report paper questionnaires, and clinic visits. Biomedical data collected during clinic visits included fasting blood samples used to assess glycosylated haemoglobin (HbA_{1c}) concentration. Informed consent was obtained prior to each data collection wave. Further details of data collection are available elsewhere (Grant et al., 2006).

7.2.3 Measures

7.2.3.1 Cardiometabolic risk

Cardiometabolic risk was expressed as HbA_{1c} concentration (%), assayed at each data collection wave from fasting blood samples collected at clinic visits. Reflective of 2-3 month time-averaged blood glucose levels, HbA_{1c} is a stable marker of glycaemic control and risk (Bennett et al., 2007). An HbA_{1c} concentration of 6.5% and above is indicative of diabetes (IEC, 2009). However, lower concentrations are monotonically related to all-cause mortality and incident CVD independently of other risk factors (blood pressure, cholesterol and lipid profiles, BMI, waist-to-hip ratio, cigarette smoking, and history of CVD) (Khaw et al., 2004).

7.2.3.2 Environmental measures

Environmental exposures were expressed within 1600m participant-centred road-network buffers, representing the distance covered by walking at a comfortable pace of 5km/hour for 19 minutes (Bohannon, 1997). Additional smaller buffer sizes (e.g., 1000m) were considered for analysis, however unstable estimates of local descriptive norms associated with smaller counts of survey participants precluded their use (discussed below). As local descriptive norms data were available for 2006-2010 only, contextual measures were expressed for 2007 to match compositional measures in time.

Local descriptive health-related norms

Geocoded population-based behavioural risk factor survey data for adults (18 years and over) were extracted from the South Australian Monitoring and Surveillance System (SAMSS) for the years 2006-2010. These data were aggregated to represent local descriptive norms.

The SAMSS survey monitors population trends in chronic diseases and risk factors (PROS, 2012). Random sampling of South Australian households from the Electronic White Pages telephone directory is used to recruit participants. The individual of any age with the most recent birthday is invited to participate. The SAMSS response rate across South Australia during 2006-2010 averaged 65% (participation rate 72.1%, 35,830 interviews conducted). Residential addresses were geocoded for 27,526 (77%) of these SAMSS participants as 16% (n=5858) did not provide consent and 7% (n=2446) lacked data necessary for geocoding. Further details of SAMSS methodology are available elsewhere (PROS, 2004).

Data from 2006 to 2010 were pooled to maximise SAMSS representation within each NWAHS participant buffer. Aggregated norms data for NWAHS buffers with fewer than 50 SAMSS participants, or less than five participants per measurement category, were not released by the data custodians in order to protect confidentiality and support the reliability of estimates. The resultant sample loss was particularly severe for the 1000m buffer size. Therefore this unit was removed from further consideration. Weightings for standardisation were not available at the level of the geographic unit used for analysis and the use of other weightings (e.g., for the Adelaide metropolitan region) may have artificially reduced or exacerbated spatial variation. Therefore, unstandardised prevalence rates were used as per the precedent of Blok and colleagues (2013).

Local descriptive body weight and physical inactivity norms were expressed as prevalence of overweight/obesity (BMI ≥ 25 kg/m^2) and insufficient physical activity (< 150 minutes of moderate intensity exercise/week) respectively (Brown et al., 2012; WHO, 2014).

Contextual measures

Contextual features assessed were POS availability and walkability, these being commonly investigated built environment measures hypothesised to support physical activity, thus conceptually matched to the compositional norms of interest to this study. The underlying rationale was that these particular measures, contextual and compositional, would be associated with change in cardiometabolic risk.

POS was defined as land parcels greater than 150m^2 available for public use, including local, linear (e.g., off-road cycling or walking path), recreational, national and conservation parks, botanic gardens, coastal areas, and outdoor sports facilities (e.g., sports fields, tennis courts).

A 2007 POS dataset was constructed using data from two sources: 1) a validated 2011 South Australian POS dataset (Daker, 2014); and 2) the 2007 South Australian Property Cadastre (South Australian Government, 2007a). POS identified from either source were subsequently validated using historical imagery from NearMap (NearMap Australia Pty Ltd), Google™ Earth and StreetView™ (Google™ Inc, USA). Non-POS land parcels were removed prior to finalising the dataset. Concordance between POS land parcels and NWAHS participant buffers was established in MapInfo using the 'intersect' function. POS availability was expressed as a proportion of buffer area (POS area/area of buffer intersected parcels).

Datasets used in construction of the walkability index were the Adelaide 2007 StreetPro™ road data (Pitney Bowes Business Insight, 2007), the 2007 Property Cadastre (South Australian Government, 2007a), and the 2007 Retail Database (South Australian Government, 2007b).

The walkability index was calculated using methods previously used in the NWAHS region (Coffee et al., 2013), based on Leslie and colleagues (2007). For each NWAHS participant buffer, walkability was calculated as the sum of deciles for four components representing connectivity (intersection density) and proximity (dwelling density, land use mix and net retail area) (Coffee, 2005; Frank et al., 2005; Leslie et al., 2007). Further detail on walkability components and their construction, is available elsewhere (Coffee et al., 2013). Walkability values range from 4 to 40, lower values representing less walkable environments.

7.2.3.3 Covariates

Individual- and area-level covariates were included in analysis models. Individual-level covariates were age, sex, employment status (full-time, part-time, or not in the work force), level of education (university graduate or not), marital status (married/de facto, or single), and smoking status (current smoker, ex-smoker, or never smoked). Covariates other than baseline age and sex were treated as time varying.

Local-area SES was represented by area-level education, extracted from the 2006 Population and Housing Census (ABS, 2006b) at the level of CDs. CDs are the smallest unit for which census data are available, each CD including an average of 220 dwellings (ABS, 2006a). CD-level data were aggregated using the weighted average of values from CDs intersected by the NWAHS participant buffers. Weights were defined based on the proportion of dwellings within a CD included within the NWAHS participant buffer:

$$BUFFER_{SES} = \sum \left[CD_{SES} \times \frac{dwellings_a}{dwellings_b} \right]$$

where $dwellings_a$ represents the number of dwellings included within a CD intersected with a buffer, and $dwellings_b$ represents the total number of dwellings within a buffer. This method assumes that the distribution of the characteristic of interest (area-level education) is evenly distributed across all dwellings. However, it is arguably an improvement over assuming that the characteristic is spread evenly across the spatial unit with no recognition of the

distribution of dwellings. To avoid multicollinearity, only one area-level SES measure was included in models.

7.2.4 Analyses

Logistic regression was used to assess predictors of attrition from the cohort following the application of analysis inclusion criteria (Table 7.1). The pattern of missingness indicated that the data did not meet the *missing completely at random* criterion. Participants who were older, not full-time employed, non- or ex-smoker, and married/de facto were more likely to attend follow up clinic visits. Insofar as the probability of non-response depended on the observed data, these measures were included in analysis models to reduce bias related to cohort attrition (Collins, Schafer & Kam, 2001; Murray & Findlay, 1988). The assumption underlying this procedure is that, conditional on the values observed, missingness occurs at random where the value of a missing observation may not in itself be informative. It is still possible, however, for results to be biased when the missing at random condition holds (Little & Rubin, 1987). Sample loss consequent to linkage with environmental exposure data was assessed using logistic regression. Individuals with linked (relative to those without linked) environmental data were older, non- or ex-smokers, employed full-time, and had higher baseline HbA_{1c}.

Associations between environmental characteristics and 10-year change in HbA_{1c} were assessed using linear multilevel models. At the first level (time level) of the model, time-specific HbA_{1c} data were regressed on time of measurement (in years) from baseline data collection. Time was measured in a continuous format from the participant's initial clinic visit. The second level (participant level) modelled the participant baseline values of, and changes in, HbA_{1c} as a function of contextual and compositional features. Random effects were specified to allow variability in baseline HbA_{1c} concentrations (intercept) and change (slope for time) across participants as a function of individual-level predictors (see below). Finally, a third level was included to account for spatial clustering within State Suburbs, with a random intercept (third level intercept) specified to account for variations in baseline values in HbA_{1c} across State Suburbs. State Suburbs are formed by aggregating CDs to align with the most recent gazetted suburb at the time of the Census (ABS, 2001).

Table 7.1 Derivation of analytic sample based on application of inclusion criteria

	n	Reason for reduced numbers
NWAHS Wave 1	4056	-
Geocoded (Baseline)	4041	15 participants with invalid residential addresses
Residing in urban area (Baseline)	3887	154 participants outside of urban area
Did not change residential address between Wave 1 and Wave 2	3322	565 participants moved between Wave 1 and Wave 2
State Suburbs with ≥10 participants	3173	149 participants resided in suburbs with ≤9 other participants
CVD/diabetes free at baseline	2621	552 participants reported CVD/diabetes at baseline
At least one set of HbA_{1c} and individual-level covariates data	2582	39 participants lacked at least one set of HbA_{1c} and individual-level covariates data
Contextual features	2213	369 participants lacked area POS availability or walkability data
Compositional features		
Prevalence of overweight/obesity (BMI≥25kg/m^2)	1890	323 participants lacked local overweight/obesity norm data
Prevalence of physical inactivity (<150 minutes/week)	1906	307 participants lacked local physical inactivity norm data

Abbreviations: BMI – body mass index; CVD – cardiovascular disease; NWAHS – North West Adelaide Health Study; POS – public open space

Models were constructed with variables included sequentially as follows: 1) compositional norm (prevalence of overweight/obesity or physical inactivity), time, and the two-way interaction of these terms (compositional norm x time); 2) context (POS availability or walkability) and the two-way interaction term for context and time; and 3) area-level education (covariate). Individual-level covariates (including predictors of cohort attrition and linkage with environmental exposure data) were included in all models. Two-way (composition norm x context) and three-way (x time) interaction terms were added to full models. Predictor x time interaction terms assess the influence of the predictor on change in HbA_{1c} over time. All analyses were conducted using SAS (version 9.4, SAS Institute Inc, Cary, North Carolina). Statistical significance was set at alpha = 0.05. Predictors were standardised prior to analyses to allow comparison of their relative effects.

7.3 Results

Inclusion criteria and consequent loss of analytic sample are shown in Table 7.1. There were 1890 and 1906 eligible NWAHS participants for analyses of local descriptive norms for overweight/obesity and physical inactivity, respectively. Table 7.2 presents characteristics of participants and the features of their environments. There were no notable differences between the two datasets. Intraclass correlations (ICC) were calculated for HbA_{1c} concentrations from covariance parameter estimates of the three-level model with no predictors. These estimates indicated moderate correlation at the individual level (repeated measures over time; ICC_{case}=0.56), and relatively low correlation at the suburb level ($ICC_{State\ Suburb}$=0.01) consistent with previously reported levels of cardiometabolic risk clustering according to geographic area (Ukoumunne et al., 1999).

Results are presented in Table 7.3 and Table 7.4. In each set of models, Model 1 included one local descriptive norm and individual covariates. Model fit (based on AIC and BIC) was improved in all series of models with the inclusion of a built environment measure (POS availability or walkability; Model 2). The inclusion of area-level education (Model 3) did not improve model fit in models including the overweight/obesity norm, nor was area-level education associated with baseline HbA_{1c}. In models including the physical inactivity norm, change in model fit with the inclusion of area-level education was inconclusive based on AIC and BIC (AIC decreased, BIC increased). Likelihood ratio tests indicated model improvement with the inclusion of area-level education ($p<0.05$) for both sets of nested models. Additionally, in Model 3 with physical inactivity norm and walkability, greater area-level education was statistically significantly associated with greater baseline HbA_{1c} concentration.

In Models 1–3, greater overweight/obesity norm and greater POS availability were each statistically significantly associated with lower baseline HbA_{1c} concentration. Physical inactivity norm and walkability were not associated with baseline HbA_{1c} concentration. Time was positively associated with HbA_{1c} concentration in all models, indicating that HbA_{1c} increased over the 10-year follow-up period. Statistically significant positive interactions with time indicate that greater overweight/obesity norm, greater physical inactivity norm, and greater POS availability were each associated with greater rates of rising HbA_{1c} over time. Greater walkability was associated with a lesser rate of increase in HbA_{1c} over time.

Regarding the composition by context interaction terms (Models 4), model fit (AIC, BIC) was not improved in three of the four sets of models. In the fourth set of models, improvement in model fit varied given inconclusive AIC and BIC values (AIC decreased, BIC increased) when analyses included two- and three-way interactions between the overweight/obesity norm, and walkability (and time). Likelihood ratio testing suggested that inclusion of these interaction terms supported an improvement in model fit (p=0.03).

There were no statistically significant two-way (composition by context) interactions related to baseline HbA_{1c} concentration. For three-way interactions (by time), only the overweight/obesity norm x walkability term was statistically significantly associated with change in HbA_{1c} over time. The effect of walkability on the relationship between local overweight/obesity prevalence and the trajectory of HbA_{1c} is shown graphically in Figure 7.2, as walkability declines the impact of the overweight/obesity norm on increasing HbA_{1c} is amplified.

Table 7.2 Individual characteristics (baseline) and environmental features (Wave 2) for participants included in analyses of norms for overweight/obesity (n=1890[a]) and physical inactivity (n=1906[a])

	Overweight/obesity norm models	Physical inactivity norm models
Individual-level characteristic	**n (%)**	**n (%)**
Sex (male)	840 (44.4%)	848 (44.5%)
Age in years [b]	49.9 (15.2)	50.0 (15.3)
Employment:		
Full-time employed	727 (38.8%)	728 (38.5%)
Part-time employed	348 (18.6%)	349 (18.5%)
Not in work-force	800 (42.7%)	815 (43.1%)
Education:		
Not university graduate	1639 (87.0%)	1655 (87.1%)
University graduate	245 (13.0%)	245 (12.9%)
Smoking status:		
Current smoker	325 (17.4%)	328 (17.4%)
Ex-smoker	631 (33.7%)	637 (33.8%)
Never smoked	915 (48.9%)	922 (48.9%)
Marital status:		
Single	663 (35.3%)	678 (35.8%)
Married/de facto	1215 (64.7%)	1216 (64.2%)
HbA_{1c} concentration (%) [b]	5.43 (0.45)	5.43 (0.45)
Environmental features	**Median (IQR)**	**Median (IQR)**
1600m buffer area (km^2)	3.90 (3.29-4.83)	3.90 (3.29-4.81)
Contextual features		
POS availability (POS area km^2/buffer km^2)	0.19 (0.09-0.30)	0.19 (0.09-0.30)
Walkability Index	24 (18-29)	24 (18-29)
Compositional features		
Overweight/obesity norm (BMI≥25kg/m^2)	62.9 (58.7-67.3)	-
$n_{(SAMSS\ participants)}$ per buffer	91 (72-116)	-
Physical inactivity norm (<150 minutes/week)	-	52.7 (48.1-57.4)
$n_{(SAMSS\ participants)}$ per buffer	-	95 (76-120)
Area education (% with university degree)	9.0 (5.9-14.7)	8.9 (5.9-14.7)

[a] n may vary due to missing values for some variables at baseline; [b] mean (SD)
Abbreviations: BMI – body mass index; IQR – interquartile range; POS – public open space

Table 7.3 Associations between local descriptive overweight/obesity norm, public open space availability, walkability, and 10-year change in HbA_{1c} concentration (n=1890) (standardised beta [β] coefficients and 95% confidence intervals [CI])

	Model 1	Model 2	Model 3	Model 4
Predictors	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**
Overweight/obesity norm & POS availability				
Time (years – not standardised)	0.0332 **** (0.0302 – 0.0363)	0.0335 **** (0.0304 – 0.0366)	0.0335 **** (0.0304 – 0.0366)	0.0333 **** (0.0302 – 0.0364)
Overweight/obesity norm	-0.0375 *** (-0.0573 – -0.0176)	-0.0364 **** (-0.0562 – -0.0166)	-0.0338 ** (-0.0573 – -0.0103)	-0.0338 ** (-0.057 – -0.0101)
Overweight/obesity norm x time	0.0080 **** (0.0049 – 0.0110)	0.0075 **** (0.0044 – 0.0106)	0.0075 **** (0.0044 – 0.0106)	0.0077 **** (0.0046 – 0.0108)
POS availability	-	-0.0223 * (-0.0428 – -0.0019)	-0.0218 * (-0.0424 – -0.0012)	-0.0216 (-0.0433 – 0.0001)
POS availability x time	-	0.0063 *** (0.0031 – 0.0095)	0.0063 *** (0.0031 – 0.0095)	0.0055 ** (0.0021 – 0.0089)
Area-level education (% university degree)	-	-	0.0049 (-0.0200 – 0.0294)	0.0050 (- 0.0196 – 0.0297)
Overweight/obesity norm x POS availability	-	-	-	-0.0011 (-0.0217 – 0.0194)
Overweight/obesity norm x POS availability x time	-	-	-	0.0024 (-0.0009 – 0.0058)
AIC	4288.0	4276.4	4278.3	4280.1
BIC	4329.5	4323.1	4327.6	4334.6
Overweight/obesity norm & walkability				
Time (years – not standardised)	0.0332 **** (0.0302 – 0.0363)	0.0342 **** (0.0311 – 0.0373)	0.0342 **** (0.0311 – 0.0373)	0.0336 **** (0.0304 – 0.0367)
Overweight/obesity norm	-0.0375 *** (-0.0573 – -0.0176)	-0.0341 ** (-0.0542 – -0.0138)	-0.0314 ** (-0.0551 – -0.0077)	-0.0314 ** (-0.0551 – -0.0078)
Overweight/obesity norm x time	0.0080 **** (0.0049 – 0.0110)	0.0066 **** (0.0035 – 0.0097)	0.0066 **** (0.0035 – 0.0097)	0.0065 **** (0.0034 – 0.0096)
Walkability	-	0.0185 (-0.0024 – 0.0395)	0.0166 (-0.0062 – 0.0394)	0.0179 (-0.0049 – 0.0408)
Walkability x time	-	-0.0077 **** (-0.0110 – -0.0045)	-0.0077 **** (-0.0110 – -0.0045)	-0.0083 **** (-0.0116 – -0.0050)
Area-level education (% university degree)	-	-	0.0056 (-0.0212 – 0.0323)	0.0059 (-0.0208 – 0.0326)
Overweight/obesity norm x walkability	-	-	-	0.0121 (-0.0069 – 0.0311)
Overweight/obesity norm x walkability x time	-	-	-	-0.0037 * (-0.0067 – -0.0006)
AIC	4288.0	4270.4	4272.3	4270.4
BIC	4329.5	4317.1	4321.6	4324.9

All models adjusted for individual-level covariates: age (at baseline), sex, marital status, education, employment status, and smoking status; *p < 0.05; **p < 0.01; ***p < 0.001; ****p< 0.0001
Abbreviations: AIC – Akaike information criterion; BIC – Bayesian information criterion; POS – public open space

Table 7.4 Associations between local descriptive physical inactivity norm, public open space availability, walkability, and 10-year change in HbA_{1c} concentration (n=1906) (standardised beta [β] coefficients and 95% confidence intervals [CI])

	Model 1	Model 2	Model 3	Model 4
Predictors	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**
Physical inactivity norm & POS availability				
Time (years – not standardised)	0.0331 **** (0.0300 – 0.0362)	0.0334 **** (0.0303 – 0.0365)	0.0334 **** (0.0304 – 0.0365)	0.0331 **** (0.0300 – 0.0362)
Physical inactivity norm	-0.0143 (-0.0346 – 0.0060)	-0.0177 (-0.0380 – 0.0026)	-0.0030 (-0.0283 – 0.0223)	-0.0035 (-0.0288 – 0.0218)
Physical inactivity norm x time	0.0057 *** (0.0027 – 0.0088)	0.0068 **** (0.0037 – 0.0099)	0.0068 **** (0.0037 – 0.0099)	0.0068 **** (0.0037 – 0.0099)
POS availability	-	-0.0269 * (-0.0477 - -0.0060)	-0.0221 * (-0.0434 – -0.0008)	-0.0247 ** (-0.0470 - -0.0025)
POS availability x time	-	0.0079 **** (0.0047 – 0.0112)	0.0079 **** (0.0047 – 0.0112)	0.0073 **** (0.0039 – 0.0108)
Area-level education (% university degree)	-	-	0.0248 (-0.0014 – 0.0509)	0.0239 (-0.0023 – 0.0500)
Physical inactivity norm x POS availability	-	-	-	-0.0094 (-0.0330 – 0.0142)
Physical inactivity norm x POS availability x time	-	-	-	-0.0021 (-0.0060 – 0.0017)
AIC	4365.6	4345.7	4344.3	4345.8
BIC	4407.1	4392.4	4393.7	4400.3
Physical inactivity norm & walkability				
Time (years – not standardised)	0.0331 **** (0.0300 – 0.0362)	0.0340 **** (0.0309 – 0.0371)	0.0340 **** (0.0309 – 0.0371)	0.0337 **** (0.0305 – 0.0368)
Physical inactivity norm	-0.0143 (-0.0346 – 0.0060)	-0.0102 (-0.0309 – 0.0105)	0.0048 (-0.0199 – 0.0294)	0.0070 (-0.0178 – 0.0319)
Physical inactivity norm x time	0.0057 *** (0.0027 – 0.0088)	0.0041 * (0.0010 – 0.0072)	0.0041 * (0.0010 – 0.0072)	0.0044 ** (0.0012 – 0.0075)
Walkability	-	0.0199 (-0.0016 – 0.0414)	0.0104 (-0.0126 – 0.0333)	0.0205 (-0.0017 – 0.0426)
Walkability x time	-	-0.0078 **** (-0.0111 - -0.0045)	-0.0078 **** (-0.0111 - -0.0045)	-0.0079 **** (-0.0112 - -0.0046)
Area-level education (% university degree)	-	-	0.0292 * (0.0015 – 0.0568)	0.0299 * (0.0026 – 0.0573)
Physical inactivity norm x walkability	-	-	-	-0.0102 (-0.0318 – 0.0114)
Physical inactivity norm x walkability x time	-	-	-	-0.0016 (-0.0051 – 0.0020)
AIC	4365.6	4348.2	4346.1	4347.8
BIC	4407.1	4394.9	4395.4	4402.3

All models adjusted for individual-level covariates: age (at baseline), sex, marital status, education, employment status, and smoking status; *p < 0.05; **p < 0.01; ***p < 0.001; ****p< 0.0001
Abbreviations: AIC – Akaike information criterion; BIC – Bayesian information criterion; POS – public open space

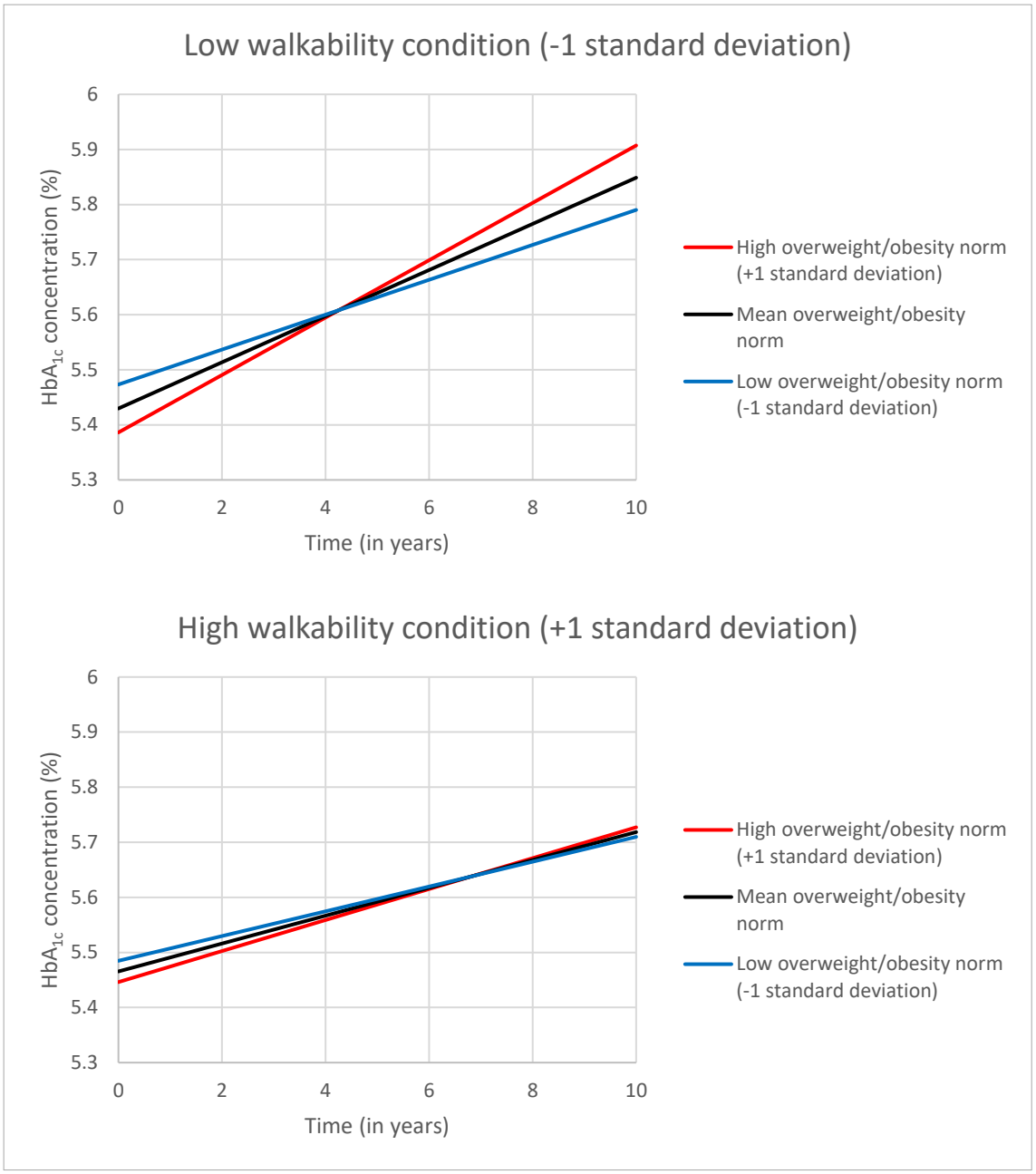

Figure 7.2 Influence of walkability on trajectories of HbA_{1c} concentration for different levels of local descriptive overweight/obesity norm

7.4 Discussion

Local descriptive health-related norms have rarely been studied in relation to cardiometabolic risk. This study found that local descriptive norms for overweight/obesity and physical inactivity predicted worsening trajectories of HbA_{1c} concentration over 10 years, accounting for individual-level demographic and smoking information (Table 7.3 and Table 7.4). These associations were evident in models that included contextual features of the built environment, and area-level education. Greater walkability was associated with a lesser increase in HbA_{1c} over 10 years, in models accounting for both individual- and area-level covariates. Unexpectedly, POS availability was positively associated with rising HbA_{1c} concentration over time. There was evidence supporting the premise that the impact of local descriptive norms on change in HbA_{1c} over time can vary according to built environment features. Greater walkability reduced the influence of the overweight/obesity norm on rate of HbA_{1c} increase. Conversely, lesser walkability exacerbated the rate of HbA_{1c} increase over time associated with greater local descriptive overweight/obesity norm.

Social network studies have reported associations between *subjective* descriptive norms and health-related outcomes. For example, a longitudinal study using data from the Framingham Heart Study found associations between social network-based norms for overweight/obesity and an individual becoming overweight/obese during the 32 years of follow-up (Christakis & Fowler, 2007). However, few studies have reported the influence of *local* descriptive norms (local prevalence of a trait or behaviour) on individual health outcomes. A longitudinal study (13 years of follow up) of Dutch adults found that the odds of an individual becoming overweight/obese increased with greater neighbourhood overweight/obesity prevalence in models adjusted for age, sex, education and neighbourhood deprivation (Blok et al., 2013). The results of the current study and that by Blok and colleagues (2013) suggest that spatial proximity, not just social proximity, is an important consideration in attempts to understand normative influences on health and health behaviour. Further research is needed in different populations and locations to support these findings.

Few investigations of local descriptive health-related norms and individual-level health outcomes have been reported. A number of studies, however, have reported on the influence of local descriptive norms on other behaviours. For example, local descriptive norms have been linked to littering and recycling behaviours (Cialdini et al., 1990; Nigbur et al., 2010).

Moreover, intervention studies have illustrated the utility of using local descriptive norms information in promoting pro-environmental behaviour. For example, one U.S. study found the provision of local descriptive norm information resulted in households significantly reducing their own energy usage (Nolan et al., 2008).

It may be possible to use similar strategies to promote individual health-related behaviour and improve population health – particularly targeting residential areas for which norms stand to be improved. Health promotion messages could include positively framed descriptive norms information. An example is, "many people walk regularly" rather than "most people don't meet physical activity recommendations". The provision of descriptive norms information alone can, however, result in what has been termed a 'boomerang effect' (Schultz et al., 2007). Descriptive norms are thought to provide a standard from which individuals do not wish to deviate (Schultz et al., 2007). Such deviance can be either above or below the norm. Therefore, the desire to conform to the norm can result in individuals decreasing participation in a desired behaviour or, conversely, increasing participation in an undesirable behaviour such as alcohol intake (Schultz et al., 2007). Inclusion of other information such as an injunctive norm message, conveying social approval or disapproval, has been shown to eliminate the boomerang effect (Schultz et al., 2007). Also, the magnitude of the norm (prevalence of overweight/obesity or physical inactivity in this study) is likely to influence the effectiveness of the norms information (Von Borgstede et al., 1999). Where a norm is expressed as the prevalence of some behaviour or condition, larger or lesser prevalence rates could exert a correspondingly greater or lesser influence on individual standards. Interventional descriptive norms information would need to be determined with the foregoing in mind. Research is needed to trial and assess the effectiveness of such interventions. Additionally, creating built environments that are supportive of physical activity may also induce the perception of a positive norm for physical activity. The provision of walking and cycling trails, and gym equipment in parks would not only enable physical activity but could help promote the perception that others are active.

In this study, positive longitudinal associations between the local descriptive overweight/obesity norm and rising HbA_{1c} differed from cross-sectional results indicating an inverse association between baseline HbA_{1c} and the overweight/obesity norm. Counter-intuitive cross-sectional results may reflect a bias due to sample attrition, loss of sample

related to inclusion criteria, or residual confounding. Sub-analyses indicated that the cross-sectional results were carried primarily by older participants, and suggest that adjusting for age did not fully remove the impact of these influences. The cross-sectional differences are clearly evident in Figure 7.2 which indicates that individuals in the low overweight/obesity norm category, despite having higher HbA_{1c} at baseline, still experienced elevations in HbA_{1c} over time, albeit to a lesser degree than higher norm categories. The longitudinal results, therefore, should be emphasised over the cross-sectional results which are probably spurious.

The current study found that POS availability was inversely associated with baseline HbA_{1c} concentration in line with the results of other cross-sectional analyses of POS availability and cardiometabolic markers. For example, a study of older Australian adults (45 and over) found greater POS availability was associated with a lesser odds of diabetes in models adjusted for individual-level sociodemographic and health measures, and area-level SES and urbanicity (Astell-Burt et al., 2014).

Despite observing an inverse relationship between POS availability and baseline HbA_{1c} our study found that POS availability was positively related to rising HbA_{1c} over 10 years. This observation diverges with a previous report by our group indicating that incident impaired fasting glucose and diabetes across a shorter 3.5 year interval (baseline to wave 2) was inversely related to the median size of available POS (Paquet et al., 2014). This prior analysis used a different definition of POS, focusing on parcels greater than $700m^2$, whereas the current study allowed for smaller parcels $150m^2$ available for public use. Further, the road-network buffers and expressions of POS differed between analyses. The previous report used a buffer of 1000m and expressed POS as the median size of available POS within the buffer, whereas the current analysis used a buffer of 1600m with POS availability expressed as POS area as a proportion of buffer intersected parcels. Such operational differences exemplify the importance of considering the form of geospatial measures used, as this can influence study results. Temporal aspects of design and analysis are also fundamentally relevant where history and maturation of cohort members could underpin the positive association observed between POS availability and change in HbA_{1c} in the current study. This could reflect a selection-maturation interaction where participants with low baseline HbA_{1c} subsequently “catch up” over 10 years due to a lagged effect with those who had higher baseline HbA_{1c}.

This study found no such contradictions with regard to walkability. Greater walkability was associated with a lesser increase in HbA_{1c} over 10 years, despite walkability not being associated with baseline HbA_{1c} concentration. No previous studies have reported on the relationship between walkability and changes in HbA_{1c} concentration; however, some longitudinal studies have assessed incidence of diabetes and pre-diabetes. For example, a Canadian study of adults (30-64 years old) in Toronto reported that low neighbourhood walkability (objectively measured) was a strong predictor of 5-year diabetes incidence in models adjusted for age and area-level income (Booth et al., 2013).

Cross-sectional studies have similarly documented associations between walkability and cardiometabolic risk. A study of Australian adults found objectively measured walkability was inversely associated with type 2 diabetes and obesity after adjusting for individual sociodemographic factors (Müller-Riemenschneider et al., 2013). The same study found no associations, however, between walkability and the cardiometabolic risk factors hypertension and hypercholesterolaemia. While cardiometabolic risk factors will cluster, it might be they have particular relationships to walkability, with glycaemic risk or disease appearing to be reasonably consistently linked to walkability in longitudinal and cross-sectional studies.

Greater walkability not only reduced the rate of increase in HbA_{1c} over time, it reduced the positive relationship between the overweight/obesity norm and increasing HbA_{1c}. For areas characterised by a greater prevalence of overweight/obesity and low walkability, the interactive effect of both influences together was a more pronounced increase in HbA_{1c} concentration over time. The authors are aware of no other study that has examined these joint influences in relation to the evolution of cardiometabolic risk.

A greater understanding of interactive effects involving different environmental risk conditions is relevant to designing and implementing contextually appropriate, *tailored* interventions to improve population health. For example, the findings of the current study suggest that improving walkability across areas with a greater overweight/obesity norm could provide greater population health benefit than improving walkability in areas with a low overweight/obesity norm. By creating environments that 1) predispose individuals towards positive health behaviours, such as through positive health-related descriptive norms, and 2) enable such health behaviours through the provision of appropriate resources (e.g., greater

walkability), the effectiveness of interventions could potentially be improved over focusing solely on one environment feature. Further exploration of the interactive effects between environmental features is warranted as a basis for locally-tailored interventions to exploit synergies between compositional population norms and contextual features that define local conditions of livings. More research is needed to address these influences in diverse populations and regions.

Developing an understanding of how local descriptive norms can be shaped is also necessary. Empirical testing of the effectiveness of health promotion strategies in relation to changing local norms is required. These strategies may need to be tailored to focus on different groups according to the current prevalence of the norm of interest. Strategies intended to influence innovators and early adopters might well differ from those for late majorities and laggards (Rogers, 2010). For example, innovators and early adopters may respond to education campaigns regarding the benefits of improving their health behaviours, the early majority may respond to the removal of barriers (such as improving the built environment), while late majorities and laggards may be more influenced by descriptive norms (Rogers, 2010).

7.4.1 Strengths and limitations

Use of a 10-year population-based, biomedical cohort with three waves of data is a strength of this study. Cardiometabolic risk was expressed using a clinical measure, HbA_{1c} concentration, an improvement over the commonly used self-reported health outcomes and their potential for self-report bias.

While the temporal ordering of the linked environmental exposures data was not ideal due to data constraints, it allowed for the testing of prospective associations, an improvement over the mostly cross-sectional studies conducted thus far on this theme. The temporal expression of environmental exposures assumes little change in spatial variation has occurred between 2000 and 2006. There was little evidence of gentrification during this time period, with 58% of study participants living in suburbs that remained in the same SES decile and 38% in suburbs that had changed by only one decile over that period. Sensitivity analyses restricted to participants residing in areas with nil or only 1 decile change found no notable differences to the main analyses. Further, whilst data pooled over 5 years might not be an ideal estimate of

the local area prevalence of overweight/obesity or physical inactivity at a point in time, the pooled data do describe the normative climate for each NWAHS participant.

Further strengths include the operationalised environmental exposures, compositional norms as well as contextual features of the built environment. The latter were represented using objective measures. POS data were extracted from a validated dataset. The walkability measure was selected based on previous use in Australia and was similar to measures used internationally such as within the International Physical Activity and Environment Network (IPEN). Local descriptive health-related norms data were derived from a separate survey, avoiding same sample bias (Diez Roux, 2007). Participant-centred road-network buffers were used to define environmental exposures (Chaix et al., 2009). Area-level education was defined using a spatial unit designed to approximate these road-network buffers. Defining environmental exposures within a range of differently sized road network buffers would have added depth to this study in capacity for sensitivity analyses. For reasons outlined, doing so was not possible. Whilst the potential influence of self-selection of respondents into residential neighbourhoods prior to baseline was not accounted for, only individuals who did not move between waves 1 and 2 were included in analyses. Further, despite efforts made to minimise confounding and reduce bias due to cohort attrition, there remains the possibility of residual confounding from unmeasured influences that could not be controlled as well as bias related to loss of analytic sample even if the missing at random criterion was upheld.

7.5 Conclusion

Compositional features beyond area-level SES, especially local descriptive norms, have rarely been investigated, most notably in relation to objective biomedical outcomes. Contextual features, such as POS availability and walkability, have been investigated more thoroughly than compositional features in this regard, but study findings vary and longitudinal studies are few.

The results of this study indicate that both contextual built environment features and compositional health-related norms predict the evolution of HbA_{1c} concentration over time in a population-based, biomedical cohort. Moreover, this study found that associations between the local descriptive norm for overweight/obesity and change in HbA_{1c} concentration differed according to the contextual built environment (walkability). Additional research in diverse locations and populations is needed to build on and extend the results of this study, particularly as a basis for ecological interventions to enable positive health-related behaviour and reduce cardiometabolic risk at the population level.

Competing interests

The authors declare that they have no competing interests.

Author contributions

SJC, MD and CP conceived and designed the study. SJC, MD, AWT, NTC, CP, and NH each made significant contributions to the acquisition and preparation of data used in the study. SJC analysed the data with input from CP and TN. SJC, MD, CP, NJH and NTC contributed to interpretation of results. SJC wrote the manuscript, and MD, CP, NJH, NTC, TN, and AWT revised it critically for important intellectual content. All authors approved the final manuscript.

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References

ABS (2001). Statistical Geography Volume 2: Census Geographic Areas Australia. Canberra: Australian Bureau of Statistics.

ABS (2003). Usual Residents Profile 2001, cat. no. 2004.0 [Online]. Canberra: Australian Bureau of Statistics.

ABS (2006a). Australian Standard Geographic Classification Vol. 2: Census Geographic Areas, Australia 2006. Canberra: Australian Bureau of Statistics.

ABS (2006b). Basic Community Profile (BCP) DataPack, cat. no. 20069.0.30.001 (Second Release). Canberra: Australian Bureau of Statistics.

Ajzen, I. (1991). The theory of planned behavior. *Organ. Behav. Hum. Dec.,* 50, 179-211.

Astell-Burt, T., Feng, X., & Kolt, G.S. (2014). Is neighborhood green space associated with a lower risk of type 2 diabetes? Evidence from 267,072 Australians. *Diabetes Care,* 37, 197-201.

Bennett, C.M., Guo, M., & Dharmage, S.C. (2007). HbA1c as a screening tool for detection of Type 2 diabetes: a systematic review. *Diabetic Med,* 24, 333-343.

Blok, D.J., de Vlas, S.J., van Empelen, P., Richardus, J.H., & van Lenthe, F.J. (2013). Changes in smoking, sports participation and overweight: does neighborhood prevalence matter? *Health Place,* 23, 33-38.

Boehmer, T.K., Hoehner, C.M., Deshpande, A.D., Brennan Ramirez, L.K., & Brownson, R.C. (2007). Perceived and observed neighborhood indicators of obesity among urban adults. *Int. J. Obesity,* 31, 968-977.

Bohannon, R.W. (1997). Comfortable and maximum walking speed of adults aged 20—79 years: reference values and determinants. *Age Ageing,* 26, 15-19.

Booth, G.L., Creatore, M.I., Moineddin, R., Gozdyra, P., Weyman, J.T., Matheson, F.I., et al. (2013). Unwalkable neighborhoods, poverty, and the risk of diabetes among recent immigrants to Canada compared with long-term residents. *Diabetes Care,* 36, 302-308.

Brown, W., Bauman, A., Bull, F., & Burton, N.W. (2012). Development of Evidence-based Physical Activity Recomendations for Adults (18-64 years). Canberra: Australian Government Department of Health.

Chaix, B., Merlo, J., Evans, D., Leal, C., & Havard, S. (2009). Neighbourhoods in eco-epidemiologic research: delimiting personal exposure areas. A response to Riva, Gauvin, Apparicio and Brodeur. *Soc. Sci. Med.,* 69, 1306-1310.

Christakis, N.A., & Fowler, J.H. (2007). The spread of obesity in a large social network over 32 years. *New Engl. J. Med.,* 357, 370-379.

Christakis, N.A., & Fowler, J.H. (2013). Social contagion theory: examining dynamic social networks and human behavior. *Stat. Med.,* 32, 556-577.

Coffee, N.T. (2005). Constructing an Objective Index of Walkability. University of Adelaide, Adelaide.

Coffee, N.T., Howard, N., Paquet, C., Hugo, G., Daniel, M. (2013). Is walkability associated with a lower cardiometabolic risk? *Health Place*, 21, 163-169.

Collins, L.M., Schafer, J.L., & Kam, C.-M. (2001). A Comparison of Inclusive and Restrictive Strategies in Modern Missing Data Procedures. *Psychol Methods,* 6, 330-351.

Cialdini, R.B., Reno, R.R., & Kallgren, C.A. (1990). A focus theory of normative conduct: recycling the concept of norms to reduce littering in public places. *J Pers Soc Psychol,* 58, 1015-1026.

Daker, M. (2014). Defining open space - not a walk in the park. School of Natural and Built Environment. Adelaide: University of South Australia.

Dal Grande, E., Taylor, A., Hurst, B., Kenny, B., & Catcheside, B. (2004). The Health Status of People Living in the South Australian Divisions of General Practice: South Australian Monitoring and Surveillance System July 2002 - December 2003. Adelaide: Population Research and Outcome Studies Unit, South Australian Department of Health.

Daniel, M., Lekkas, P., Cargo, M., Stankov, I., Brown, A. (2011). Environmental risk conditions and pathways to cardiometabolic diseases in Indigenous populations. *Annu. Rev. Publ. Health*, 32, 327-347.

Daniel, M., Moore, S., Kestens, Y. (2008). Framing the biosocial pathways underlying associations between place and cardiometabolic disease. *Health Place*, 14, 117-132.

Diez Roux, A.V. (2007). Neighborhoods and health: where are we and where do we go from here? *Revue d'Epidémiologie et de Santé Publique,* 55, 13-21.

Frank, L.D., Schmid, T.L., Sallis, J.F., Chapman, J., & Saelens, B.E. (2005). Linking objectively measured physical activity with objectively measured urban form: findings from SMARTRAQ. *Am. J. Prev. Med.,* 28, 117-125.

Granovetter, M. (1978). Threshold models of collective behavior. *American Journal of Sociology,* 83, 1420-1443.

Grant, J., Chittleborough, C., Taylor, A., Dal Grande, E., Wilson, D., Phillips, P., et al. (2006). The North West Adelaide Health Study: detailed methods and baseline segmentation of a cohort for chronic diseases. *Epidemiological Perspectives and Innovations,* 3.

IEC (2009). International Expert Committee Report on the role of the A1C assay in the diagnosis of diabetes. *Diabetes Care,* 32, 1327-1334.

Khaw, K.T., Wareham, N., Bingham, S., Luben, R., Welch, A., & Day, N. (2004). Association of hemoglobin A1c with cardiovascular disease and mortality in adults: the European Prospective Investigation into Cancer in Norfolk. *Ann Intern Med,* 141, 413-420.

Kormos, C., Gifford, R., & Brown, E. (2015). The influence of descriptive social norm information on sustainable transportation behavior: a field experiment. *Environment and Behavior,* 47, 479-501.

Leal, C., & Chaix, B. (2011). The influence of geographic life environments on cardiometabolic risk factors: a systematic review, a methodological assessment and a research agenda. *Obes. Rev.,* 12, 217-230.

Leslie, E., Coffee, N., Frank, L., Owen, N., Bauman, A., & Hugo, G. (2007). Walkability of local communities: using geographic information systems to objectively assess relevant environmental attributes. *Health Place,* 13, 111-122.

Little, R.J.A., & Rubin, D.B. (1987). *Statistical Analysis with Missing Data*. New York: John Wiley & Sons.

Müller-Riemenschneider, F., Pereira, G., Villanueva, K., Christian, H., Knuiman, M., Giles-Corti, B., et al. (2013). Neighborhood walkability and cardiometabolic risk factors in Australian adults: an observational study. *BMC Public Health,* 13, 1-9.

Murray, G.D., & Findlay, J.G. (1988). Correcting for the bias caused by drop-outs in hypertension trials. *Stat. Med.,* 7, 941-946.

Nigbur, D., Lyons, E., & Uzzell, D. (2010). Attitudes, norms, identity and environmental behaviour: using an expanded theory of planned behaviour to predict participation in a kerbside recycling programme. *Br J Soc Psychol,* 49, 259-284.

Nolan, J.M., Schultz, P.W., Cialdini, R.B., Goldstein, N.J., & Griskevicius, V. (2008). Normative social influence is underdetected. *Pers Soc Psychol B,* 34, 913-923.

Paquet, C., Coffee, N.T., Haren, M.T., Howard, N.J., Adams, R.J., Taylor, A.W., et al. (2014). Food environment, walkability, and public open spaces are associated with incident development of cardio-metabolic risk factors in a biomedical cohort. *Health Place*, 28, 173-176.

Pitney Bowes Business Insight (2007). Adelaide 2007 StreetPro and Roads. Pitney Bowes Business Insight.

PROS. (2004). South Australian Monitoring and Surveillance System: Survey Methodology. SAMSS Technical Paper Series No. 1/04, August 2004. Adelaide: Population Research and Outcome Studies and SA Department of Health.

PROS. (2012). South Australian Monitoring and Surveillance System (SAMSS). Adelaide: Population Research and Outcome Studies.

Richard, L., Gauvin, L., & Raine, K. (2011). Ecological models revisited: their uses and evolution in health promotion over two decades. *Annu. Rev. Publ. Health,* 32, 307-326.

Rivis, A., & Sheeran, P. (2003). Descriptive norms as an additional predictor in the theory of planned behaviour: a meta-analysis. *Curr. Psychol.,* 22, 218-233.

Rogers, E.M. (2010). *Diffusion of innovations*: Simon and Schuster.

Schultz, P.W., Nolan, J.M., Cialdini, R.B., Goldstein, N.J., & Griskevicius, V. (2007). The constructive, destructive, and reconstructive power of social norms. *Psychol Sci,* 18, 429-434.

South Australian Department of Health. (2005). HOS: Self Reported Prevalence of Obesity in the SA Health Regions. Adelaide: Population Research and Outcome Studies Unit, South Australian Department of Health.

South Australian Government (2007a). South Australian Property Cadastre, Department of Planning, Transport and Infrastructure (DPTI); Adelaide, South Australia.

South Australian Government (2007b). South Australian Retail Database, Department of Planning, Transport and Infrastructure (DPTI); Adelaide, South Australia.

Ukoumunne, O., Gulliford, M., Chinn, S., Sterne, J., & Burney, P. (1999). Methods for evaluating area-wide and organisation-based interventions in health and health care: a systematic review. *Health Technology Assessment,* 3, 98.

Von Borgstede, C., Dahlstrand, U., & Biel, A. (1999). From ought to is: moral norms in large-scale social dilemmas. *Goteberg Psychological Reports,* 5, 1-17.

WHO. (2014). Global Database on Body Mass Index: An Interactive Surveillance Tool for Monitoring Nutrition Transition. Geneva: World Health Organization,.

Wilcox, S., Castro, C., King, A.C., Housemann, R., & Brownson, R.C. (2000). Determinants of leisure time physical activity in rural compared with urban older and ethnically diverse women in the United States. *J. Epidemiol. Community Health,* 54, 667-672.

Chapter 8

Results 3

8 Results 3

The third of the published manuscripts is presented here in Chapter 8. This manuscript attends Objectives 2 and 3 within the food environment. Authorship contributions and copyright information is provided in Appendix I. The article reference is:

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RESEARCH ARTICLE Open Access

Local descriptive body weight and dietary norms, food availability, and 10-year change in glycosylated haemoglobin in an Australian population-based biomedical cohort

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Abstract

Background: Individual-level health outcomes are shaped by environmental risk conditions. Norms figure prominently in socio-behavioural theories yet spatial variations in health-related norms have rarely been investigated as environmental risk conditions. This study assessed: 1) the contributions of local descriptive norms for overweight/obesity and dietary behaviour to 10-year change in glycosylated haemoglobin (HbA_{1c}), accounting for food resource availability; and 2) whether associations between local descriptive norms and HbA_{1c} were moderated by food resource availability.

Methods: HbA_{1c}, representing cardiometabolic risk, was measured three times over 10 years for a population-based biomedical cohort of adults in Adelaide, South Australia. Residential environmental exposures were defined using 1600 m participant-centred road-network buffers. Local descriptive norms for overweight/obesity and insufficient fruit intake (proportion of residents with BMI ≥ 25 kg/m^2 [$n = 1890$] or fruit intake of <2 serves/day [$n = 1945$], respectively) were aggregated from responses to a separate geocoded population survey. Fast-food and healthful food resource availability (counts) were extracted from a retail database.
Separate sets of multilevel models included different predictors, one local descriptive norm and either fast-food or healthful food resource availability, with area-level education and individual-level covariates (age, sex, employment status, education, marital status, and smoking status). Interactions between local descriptive norms and food resource availability were tested.

Results: HbA_{1c} concentration rose over time. Local descriptive norms for overweight/obesity and insufficient fruit intake predicted greater rates of increase in HbA_{1c}. Neither fast-food nor healthful food resource availability were associated with change in HbA_{1c}. Greater healthful food resource availability reduced the rate of increase in HbA_{1c} concentration attributed to the overweight/obesity norm.
(Continued on next page)

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Abstract

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Separate sets of multilevel models included different predictors, one local descriptive norm and either fast-food or healthful food resource availability, with area-level education and individual-level covariates (age, sex, employment status, education, marital status, and smoking status). Interactions between local descriptive norms and food resource availability were tested.

Results: HbA_{1c} concentration rose over time. Local descriptive norms for overweight/obesity and insufficient fruit intake predicted greater rates of increase in HbA_{1c}. Neither fast-food nor healthful food resource availability were associated with change in HbA_{1c}. Greater healthful food resource availability reduced the rate of increase in HbA_{1c} concentration attributed to the overweight/obesity norm.

Conclusions: Local descriptive health-related norms, not food resource availability, predicted 10-year change in HbA_{1c}. Null findings for food resource availability may reflect a sufficiency or minimum threshold level of resources such that availability poses no barrier to obtaining

healthful or unhealthful foods for this region. However, the influence of local descriptive norms varied according to food resource availability in effects on HbA_{1c}. Local descriptive health-related norms have received little attention thus far but are important influences on individual cardiometabolic risk. Further research is needed to explore how local descriptive norms contribute to chronic disease risk and outcomes.

Keywords

cardiometabolic risk, food environment, built environment, descriptive norms, multilevel models

8.1 Background

Public health interventions commonly focus on modifiable individual-level risk factors such as dietary behaviour. However, individual-level risk factors are themselves shaped by environmental risk conditions, that is, properties of environmental living conditions that exacerbate a vulnerability to disease for the individuals exposed to those places [1]. Individual-level health behaviours, such as dietary choices, are one possible pathway through which local environments may influence health outcomes such as cardiometabolic risk [2]. For example, fast food intake may be influenced by the number of fast-food outlets in an individual's residential area [3].

Environmental features can be contextual (i.e., features of areas) or compositional (i.e., aggregated characteristics of people residing within areas) [1,4]. Both contextual and compositional features are associated with cardiometabolic risk. A comprehensive review concluded there were reasonably consistent associations reported between accessibility to a supermarket and lower body weight, and between convenience store and fast-food outlet accessibility and higher body weight [5], higher body weight being a cardiometabolic risk factor. Some studies, however, have not observed any relationship between cardiometabolic risk and features of the food environment. Others have observed counterintuitive associations. One US study among low-income women reported no associations between body mass index (BMI) or cardiovascular disease (CVD) risk and the density of grocery stores, fast-food outlets, restaurants, or minimarts [6]. Similarly, a multi-ethnic study of pregnant women in the UK observed no associations between fast-food availability (count of outlets) or accessibility (distance to nearest outlet) and BMI or obesity for non-South Asian pregnant women [7]. For South Asian pregnant women, the same study reported an unexpected negative association between fast-food availability and accessibility and BMI and obesity [7]. Explanations for null or unexpected observations need to reach beyond demographic attributions such as ethnicity and socioeconomic status (SES). It is possible that additional, broader factors, not accounted for by statistical adjustments for SES, such as norms, could shape the nature of relationships between food resources and health outcomes.

Numerous studies have investigated whether contextual features of local environments (e.g., fast-food outlets) are related to cardiometabolic risk, particularly body weight. Fewer studies have assessed the relationships between cardiometabolic risk and compositional features of

local environments, beyond area-level SES. Associations between area-level SES and cardiometabolic risk are now very well established [5]. What remains to be far better investigated are the aggregated characteristics of people *beyond* area-level SES, for example, health-related norms, as they vary geographically. Local descriptive health-related norms may be important factors shaping cardiometabolic risk and disease through their effects on collective lifestyles and behaviour.

Though norms feature prominently in behavioural theories, for example the Theory of Planned Behaviour [8], norms are not always well defined within research. Social norms can be differentiated into *injunctive* and *descriptive* norms [9]. *Injunctive norms* are 'shared rules of conduct', that is, what *ought* to be done, while *descriptive norms* are what most people actually do [9]. Injunctive and descriptive norms are likely to influence individuals through different motivational processes [9,10].

Descriptive norms can be further differentiated into *subjective* and *local* descriptive norms [11,12]. *Subjective descriptive norms* refers to what friends and family typically do. In contrast, *local descriptive norms* are what people sharing the same spatial setting, such as a work-place or residential area, typically do. This is regardless of any emotional connection, or lack thereof, between individuals within the setting [11-13]. Local descriptive norms have been associated with littering and recycling behaviours [9,14]. While subjective descriptive norms, such as smoking behaviour, have been explored within social networks [15], local descriptive norms have rarely been examined in relation to health outcomes.

A longitudinal study (involving 13 years of follow up) by Blok and colleagues [16], found neighbourhood prevalence of overweight/obesity predicted normal weight individuals becoming overweight/obese after accounting for individual factors and neighbourhood SES. Unfortunately, the study did not account for contextual features of the local environment, such as food availability, which may account for both prevalence of overweight/obesity and change in individual-level BMI. A recent longitudinal study using the same cohort reported on here accounted for contextual features of the physical activity environment, finding that local descriptive norms for overweight/obesity and physical inactivity predicted rising HbA_{1c} concentrations over time [17].

Local descriptive health-related norms may be important influences on clinical outcomes by predisposing individuals towards or against particular health behaviours. It is important to empirically assess the influence of such norms on individual-level health outcomes, ideally while accounting for potential confounders such as availability of health-related resources. Furthermore, while local descriptive health-related norms may act as predisposing factors for health-related behaviours, the availability of contextual resources may enable (or inhibit) such behaviour. Thus the availability of health-related resources may modify associations between local descriptive health-related norms and health outcomes that are a function of behaviour. For example, associations between a local descriptive norm for overweight/obesity and the development of cardiometabolic risk in individuals may be more pronounced in areas with greater, as opposed to lesser, fast-food availability.

Few studies have assessed contextual and compositional interaction effects in relation to important public health issues such as the rising level of cardiometabolic risk. Specifically, no study published thus far has investigated whether cardiometabolic risk is related to spatial variation in local-area norms for body weight and dietary behaviour while accounting for the built food environment, and whether any such relationship varies with food resource availability. This study assessed in a population-based biomedical cohort: 1) the influence of local descriptive norms for body weight and dietary behaviour on 10-year change in HbA_{1c} (a marker of cardiometabolic risk); and 2) whether associations between change in HbA_{1c} and local descriptive norms for body weight and dietary behaviour varied according to food resource availability.

8.2 Methods

This study used an observational design incorporating data from a prospective biomedical cohort linked with other data sets utilising a Geographic Information System. The study was part of the Place and Metabolic Syndrome (PAMS) Project which aimed to assess the influence of social and built environmental factors on the evolution of cardiometabolic risk. The PAMS Project received ethical approval from the University of South Australia, Central Northern Adelaide Health Service, Queen Elizabeth Hospital, and South Australian Department for Health and Ageing Human Research Ethics Committees.

8.2.1 Study Area

The baseline study area consisted of the northern and western regions of Adelaide (Figure 8.1), the capital city of South Australia. These regions accounted for 38% of the city's 1.1 million population in 2001 [18,19] and are of particular interest due to elevated cardiometabolic risk relative to other areas [20,21].

Associations between environments, health behaviours and outcomes may differ between urban and rural regions [22]. This study was therefore limited to urban areas only, defined as Census Collection Districts (CDs) with a population density of >200 persons per hectare [19].

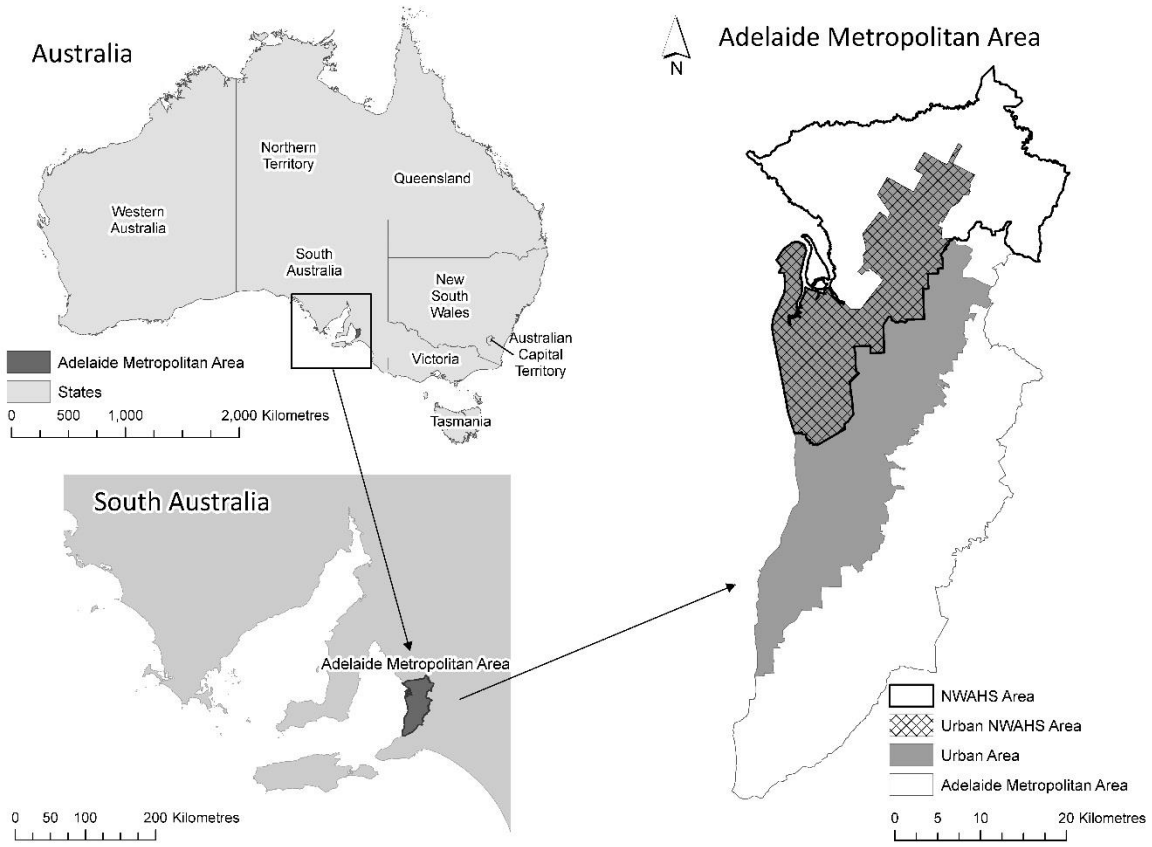

Figure 8.1 Study area – North-western region of Adelaide (urban area)
(Reprinted from Carroll et al 2016, with permission from Elsevier)

8.2.2 Participants

Individual-level data were sourced from the North West Adelaide Health Study (NWAHS), a 10-year biomedical cohort incorporating three waves of data collection, Wave 1 (2000-03), Wave 2 (2005-06), and Wave 3 (2008-10). The NWAHS investigated the prevalence of chronic conditions, including diabetes and cardiovascular disease, and their associated risk factors [23]. Households identified as within the study region by postcode were randomly selected from the Electronic White Pages telephone directory, and the person aged 18 years or over with the most recent birthday invited to participate in the study. Each NWAHS wave involved the collection of standardised measures using Computer-Assisted Telephone Interviews, self-report paper questionnaires, and clinic visits. Fasting blood samples were collected during the clinic visits and used to assess glycosylated haemoglobin (HbA_{1c}) concentration. Written informed consent was obtained prior to each wave of data collection. Georeference points, made from participant residential addresses at each wave, enabled data linkage with other spatial datasets.

To retain cohort study participants, a multi-strategy approach was employed including consistent use of study promotional materials, newsletters and birthday cards, tracking via White Pages telephone directory and State Electoral Roll [23]. Of the 4056 Wave 1 participants, 3205 (79.0% of baseline sample) attended the Wave 2 clinic assessment and 2487 (77.6% of Wave 2 sample; 61.3% of baseline sample) attended the clinic at Wave 3. The baseline NWAHS sample was not statistically significantly different to the Adelaide metropolitan population [24] by sex, education or household income. However, older individuals (≥ 45 years) were over-represented in the baseline sample. Further information on recruitment and cohort profile has previously been published [23,25].

8.2.3 Measures

8.2.3.1 Cardiometabolic risk (outcome measure)

Glycosylated haemoglobin (HbA_{1c}) concentration (%), assayed at each wave, was used to represent cardiometabolic risk. HbA_{1c} is a stable marker of glycaemic control and thus risk, reflecting 2-3 month time-averaged blood glucose levels [26]. Concentrations 6.5% or greater are indicative of diabetes [27]. However, the relationship between HbA_{1c} and cardiovascular disease (CVD) is continuous and lacking an obvious risk threshold [28].

8.2.3.2 Environmental measures

Environmental exposures were expressed within spatial units defined as participant-centred road-network buffers set to 1600m (1 mile). This distance can be covered by an average adult walking at a comfortable pace of around 5km/hour for approximately 20 minutes [29]. The 1600m buffer distance has previously been used in similar studies [e.g., 30,31,32] allowing for comparison of findings across studies. Smaller buffers of 1000m were also considered but dropped due to unstable estimates of local descriptive norms associated with small counts of survey participants within buffers (see below).

Geocoded data for constructing local descriptive norms were not available prior to 2006. To temporally match data for local descriptive norms, other environmental exposures were expressed for the year 2007.

Local descriptive health-related norms

Local descriptive norms for overweight/obesity and insufficient fruit intake were respectively expressed as local prevalence of overweight/obesity (proportion of South Australian

Monitoring and Surveillance System [SAMSS] participants per buffer classified as having a BMI ≥ 25kg/m^2) and insufficient fruit intake (proportion of SAMSS participants per buffer not meeting fruit intake recommendations), based on health recommendations of two or more serves per day [33,34].

Local descriptive norms were aggregated from geocoded individual-level survey response data (adults 18 years and older), extracted from the SAMSS for the years 2006-2010. Processing of individual-level SAMSS data was performed by the data custodians to protect the confidentiality of SAMSS participants.

The SAMSS survey for which details are published elsewhere, monitors population trends in chronic diseases and risk factors [35,36]. SAMSS participants are recruited annually across all of South Australia by simple random sampling of households from the Electronic White Pages telephone directory. The individual, of any age, with the most recent birthday is invited to participate. Overall, the response rate for SAMSS contacts during 2006-2010 was 65% with 35,830 interviews conducted across South Australia. Of the 8355 SAMSS participants interviewed during 2006-2010, 18 years and over residing within the NWAHS region, 6860 participant records were geocoded (82%); 1439 participants did not provide consent (17%) and 56 (<1%) could not be geocoded.

To maximise SAMSS participant representation within each NWAHS participant buffer, SAMSS data were pooled across survey years 2006 to 2010. To protect confidentiality and support the reliability of estimates, aggregated norms data for NWAHS buffers with fewer than 50 SAMSS participants, or less than five participants per measurement category, were not released by the data custodians. Consequently sample loss occurred which was particularly severe at the 1000m buffer size and hence this unit was not considered further. Unstandardised prevalence rates were used following the precedent of Blok and colleagues [16]. Appropriate weightings for standardisation were unavailable at the level of the geographic buffers used, and the use of other weightings (e.g., for the Adelaide metropolitan region) may artificially reduce or inflate spatial variation.

Contextual features

Contextual data were extracted from the 2007 South Australian Retail Database [37]. The database catalogues shops, with information including shop location, retail activity type, and

shop floor-space. Retail activity type is coded based on predominant retail activities [38]. Contextual food environment data were extracted according to these retail codes.

Food resources were classified by the authors based on these retail codes, using classifications designed by a dietician for use in a previous Australian study [39]. Fast-food outlets were defined as major fast-food franchises (e.g., McDonalds©) and independent fast-food take-away stores (e.g., fish and chips). Healthful food resources were defined as greengrocers, butchers, supermarkets (with > 200m^2 floor space), and health food shops. Food outlets selling a mix of healthful and unhealthful foods, with neither food group being obviously predominant (e.g., sandwich and lunch bars, bakeries, and restaurants other than those identified as fast food), were excluded from classification.

Road-network distance from NWAHS participants' residence to food resources was calculated using Network Spatial Analyst in ArcGIS (version 9.3.1, ESRI, Redlands, California). Healthful food resources and fast-food outlets identified within 1600m of participant residences were then summed according to type. Density measures (count/area of buffer intersected parcels in km^2) were calculated in addition to counts.

8.2.3.3 Covariates

Individual- and area-level covariates were included in analytic models. Predictors of NWAHS cohort attrition were assessed using logistic regression within the analytic sample (i.e., after application of inclusion criteria as listed in Table 8.1). The pattern of missingness did not meet the *missing completely at random* criterion. As participants who were younger, not in the work-force, currently a smoker or ex-smoker, and not married (or de facto) were more likely to have missing HbA_{1c} information at follow ups, these measures were included in statistical models to satisfy the analytic criterion of *missing at random* [40].

Therefore individual-level covariates included age, sex, employment status (full-time, part-time, or not in the work force), level of education (university graduate or not), marital status (married/de facto, or single), and smoking status (current smoker, ex-smoker, or never smoked). Covariates other than baseline age and sex were treated as time varying.

Table 8.1 Loss of analytic sample due to application of inclusion criteria

	n	Reason for reduced numbers
NWAHS Wave 1	4056	-
Geocoded (Baseline)	4041	15 participants with invalid residential addresses
Residing in urban area (Baseline)	3887	154 participants outside of urban area
Did not change residential address between Wave 1 and Wave 2	3322	565 participants moved between Wave 1 and Wave 2
State Suburbs with ≥ 10 participants	3173	149 participants resided in suburbs with ≤ 9 other participants
CVD/diabetes free at baseline	2621	552 participants reported CVD/diabetes at baseline
At least one set of HbA_{1c} and individual-level covariates data	2582	39 participants did not have at least one set of complete HbA_{1c} and individual-level covariates data
Contextual features data	2213	369 participants did not have fast-food outlet or healthful food resource availability data at Wave 2
Compositional norms data		
Prevalence of overweight/obesity (BMI ≥ 25kg/m^2)	1890	323 participants lacked local descriptive overweight/obesity data
Prevalence of insufficient fruit intake (< 2 serves daily)	1945	268 participants lacked local descriptive fruit intake data

Area-level education (proportion with a university degree) was selected to represent area-level SES. The use of area-level education allows interpretation of specific area-level SES relations with health outcomes (i.e., change in HbA_{1c}) and comparisons with studies similarly using education to express area-level SES. Education data were extracted from the 2006 Population and Housing Census [41] at the level of CDs and further aggregated using the weighted average of values from CDs intersected by the NWAHS participant buffers. CDs, the smallest unit for which census data are available, include an average of 220 dwellings [42]. Weights were defined based on the proportion of dwellings within a CD included within the NWAHS participant buffer:

$$BUFFER_{SES} = \sum \left[CD_{SES} \times \frac{dwellings_a}{dwellings_b} \right]$$

where $dwellings_a$ represents the number of dwellings included within a CD intersected with a buffer, and $dwellings_b$ represents the total number of dwellings within a buffer. Though assuming that the distribution of the characteristic of interest (area-level education) is evenly distributed across all dwellings, this method is an improvement over assuming that the

characteristic is evenly spread across the spatial unit with no recognition of the distribution of dwellings.

8.2.4 Analyses

Linear multilevel models (three levels), assessed associations between environmental features and 10-year change in HbA_{1c}. Level one of the model (time) regressed time-specific HbA_{1c} data on time of measurement (in years) from baseline data collection. As data collection between participant waves was unevenly spaced, with slightly different years possible within each wave, time was expressed in a continuous format from the participant's first clinic visit. Level two (participant level) modelled associations between environmental exposures and participant baseline values of, and changes in, HbA_{1c}. Included random effects allowed variation in baseline HbA_{1c} (intercept) and HbA_{1c} change (slope for time) between participants. Lastly, level three accounted for spatial clustering within State Suburbs, with a random intercept specified to allow for variations in baseline HbA_{1c} across State Suburbs. State Suburbs are formed by aggregating CDs to align with the most recent gazetted suburb at the time of the Census [19].

Four separate sets of models were constructed, with individual-level covariates included in all models. Predictor variables were added sequentially: 1) compositional norm (prevalence of overweight/obesity or insufficient fruit intake), time, and the two-way interaction between these terms; 2) context (fast food or healthful food availability), and the two-way interaction term (context x time); and 3) area-level education (covariate). Interaction terms for predictors and time (e.g., compositional norm x time) assessed the influence of the predictor (compositional norm) on *change* in HbA_{1c} over time. Additional two-way (compositional norm x context) and three-way (compositional norm x context x time) interaction terms were included in full models to test for interactions between environmental predictors in relation to baseline HbA_{1c} and *change* in HbA_{1c} respectively.

Environmental measures were standardised prior to analyses to allow comparison of their relative effects. All analyses were conducted using SAS (version 9.4, SAS Institute Inc, Cary, North Carolina). Statistical significance was set at alpha = 0.05.

8.3 Results

Table 8.1 outlines sample loss due to analysis inclusion criteria. Participants who moved between waves 1 and 2 were excluded from analyses. The two analytic samples contained were 1890 and 1945 eligible NWAHS participants with local descriptive norms data for overweight/obesity and insufficient fruit intake, respectively. Participant characteristics and environmental features are summarised in Table 8.2. There were no notable differences between the two analysis samples. The majority (90.2%) of eligible participants were born in Australia, New Zealand or Western Europe, and the median length of follow-up was 6.84 years for both samples.

Intraclass correlations (ICC), describing the degree of similarity (or homogeneity) of the observed response within a given unit of analysis (i.e., HbA_{1c} concentration across waves for a participant) or cluster (i.e., State Suburb) were calculated from covariance parameter estimates of the three-level model with no predictors [43]. These ICCs indicated moderate correlation of HbA_{1c} at the individual level (repeated measures over time; $ICC_{participants} = 0.57$) and relatively low correlation at the suburb level ($ICC_{State\ Suburb} = 0.01$) consistent with previously reported levels of cardiometabolic risk clustering according to geographic area [44].

Table 8.2 Individual characteristics and environmental features for the analytic samples

Individual-level characteristic (baseline)	Overweight/obesity norm models (n=1890)[a] n (%)	Insufficient fruit intake norm models (n=1945)[a] n (%)
Length of follow-up (years) [b]	6.84 (4.59-8.36)	6.84 (4.54-8.36)
Sex (male)	840 (44.4%)	864 (44.4%)
Age in years [c]	49.9 (15.2)	50.0 (15.2)
Ethnicity:		
Born in Australia/New Zealand/Western Europe	1698 (90.2%)	1748 (90.2%)
Employment:		
Full-time employed	727 (38.8%)	747 (38.7%)
Part-time employed	348 (18.6%)	357 (18.5%)
Not in work-force	800 (42.7%)	826 (42.8%)
Education:		
Not university graduate	1639 (87.0%)	1689 (87.1%)
University graduate	245 (13.0%)	250 (12.9%)
Smoking status:		
Current smoker	325 (17.4%)	335 (17.4%)
Ex-smoker	631 (33.7%)	650 (33.8%)
Never smoked	915 (48.9%)	940 (48.8%)
Marital status:		
Single	663 (35.3%)	686 (35.5%)
Married/de facto	1215 (64.7%)	1247 (64.5%)
HbA_{1c} concentration (%) [c]	5.43 (0.45)	5.43 (0.45)
Environmental features	**Mean (SD)**	**Mean (SD)**
1600m buffer area (km^2) [b]	3.90 (3.29-4.83)	3.90 (3.29-4.84)
Contextual features		
Fast-food outlets (count)	5.5 (3.9)	5.5 (3.9)
Fast-food outlet density (count/buffer km^2)	1.67 (1.26)	1.65 (1.26)
Healthful food resources (count)	4.1 (3.4)	4.0 (3.4)
Healthful food resource density (count/buffer km^2)	1.01 (0.89)	1.01 (0.89)
Compositional features		
Overweight/obesity norm (BMI ≥ 25kg/m^2)	62.97% (2.21%)	-
$n_{(SAMSS\ participants)}$ per buffer [b]	91 (72-116)	-
Insufficient fruit intake norm (< 2 serves daily)	-	53.82% (6.55%)
$n_{(SAMSS\ participants)}$ per buffer [b]	-	96 (76-122)
Area-level education (% with university degree)	10.19% (5.17)	10.09% (5.18)

[a] total *n* may vary due to missing values for some variables at baseline; [b] median (IQR); [c] mean/SD

Table 8.3 and Table 8.4 present the results of the four sets of multilevel models and adjusted ICCs. As environmental exposure measures (including area-level education) were standardised prior to analyses, the reported beta coefficients reflect change in HbA_{1c} concentration per one standard deviation (SD) change in the environmental exposure predictor. Means and SDs of environmental measures are provided in Table 8.2. Model 1 in each set included time, one local descriptive (either overweight/obesity or insufficient fruit intake) and individual-level covariates. Model fit (based on AIC and BIC) did not improve in any of the four sets of models with the inclusion of measures of food resource availability (neither fast food nor healthful food resources; Model 2). Similarly, the inclusion of area-level education at Model 3 did not improve model fit in sets of models including the overweight/obesity norm (Table 8.3). However, model fit did improve with inclusion of area-level education in models with the insufficient fruit intake norm (Table 8.4, Model 3), and area-level education was statistically significantly positively associated with baseline HbA_{1c}. Lastly, the inclusion of the environmental exposures interaction term in model 4 did not improve model fit in three of the four sets of models. In the fourth set, the inclusion of an interaction term between the featured environmental predictors, namely overweight/obesity norm and healthful food resources, improved model fit.

In Models 1–3, lesser overweight/obesity norm was statistically significantly associated with greater baseline HbA_{1c} concentration (β = -0.03 to -0.04 depending on model; i.e., a 2.21% [1SD] increment in overweight/obesity prevalence was associated with a -0.03% to -0.04% lower HbA_{1c} concentration). Insufficient fruit intake norm, fast-food outlets, and healthful food resources were not associated with baseline HbA_{1c}. HbA_{1c} increased over the 10-year follow-up period (time was statistically significantly positively associated with HbA_{1c} concentration in all models) with an increase in HbA_{1c} concentration of 0.03% per year. Statistically significant positive time x norm interactions indicate that greater prevalences for the overweight/obesity norm, and greater insufficient fruit intake norm, were each associated with greater rates of rising HbA_{1c} over time (e.g., in model 3 with fast food availability: overweight/obesity norm x time β = 0.008 indicating that a 2.21% [1SD] increment in overweight/obesity prevalence was associated with a further 0.008% increase in HbA_{1c} per year).

Fast-food outlets and healthful food resources were not associated with change in HbA_{1c} over time. There were no statistically significant two-way (local descriptive norm and food resource availability) interactions related to baseline HbA_{1c} concentration.

The three-way interaction of the overweight/obesity norm x healthful food resource x time was statistically significantly associated with HbA_{1c} (β = -0.0057 [95% CI -0.0092 to -0.0022], p=0.001). The effect of healthful food resource availability on the relationship between local descriptive overweight/obesity norm and the trajectory of HbA_{1c} is shown graphically in Figure 8.2. The figure shows that greater healthful food resource availability reduced the impact of the overweight/obesity norm on increasing HbA_{1c} concentration. Models including the food environment measures as density rather than count measures found similar results (not reported here).

Table 8.3 Associations between local descriptive overweight/obesity norm, food resource availability, and 10-year change in HbA_{1c}

	Model 1	Model 2	Model 3	Model 4
Predictors	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**
Overweight/obesity norm and fast-food outlet availability				
Time (in years – not standardised)	0.0332 **** (0.0301 – 0.0363)	0.0332 **** (0.0301 – 0.0636)	0.0332 **** (0.0301 – 0.0363)	0.0331 **** (0.0300 – 0.0362)
Overweight/obesity norm	-0.0376 *** (-0.0575 to -0.0178)	-0.0382 *** (-0.0580 - -0.0183)	-0.0335 ** (-0.0571 to -0.0100)	-0.0334 ** (-0.0569 to -0.0098)
Overweight/obesity norm x time	0.0080 *** (0.0049 – 0.0110)	0.0080 **** (0.0049 – 0.0111)	0.0080 **** (0.0049 – 0.0111)	0.0080 **** (0.0049 – 0.0111)
Fast-food outlets	-	-0.0082 (-0.0281 – 0.0116)	-0.0096 (-0.0298 – 0.0106)	-0.0094 (-0.0296 – 0.0108)
Fast-food outlets x time	-	0.0002 (-0.0030 – 0.0034)	0.0002 (-0.0030 – 0.0034)	0.0003 (-0.0029 – 0.0035)
Area-level education (% university degree)	-	-	0.0090 (-0.0158 – 0.0337)	0.0089 (-0.0158 – 0.0336)
Overweight/obesity norm x fast-food outlets	-	-	-	-0.0021 (-0.0228 – 0.0186)
Overweight/obesity norm x fast-food outlets x time	-	-	-	-0.0021 (-0.0055 – 0.0014)
AIC	4286.0	4289.3	4290.8	4293.0
BIC	4327.5	4336.0	4340.1	4347.5
Adjusted $ICC_{(participants)}$	0.60	0.60	0.60	0.60
Adjusted $ICC_{(State\ Suburb)}$	0.01	0.01	0.01	0.01
Overweight/obesity norm and healthful food resource availability				
Time (in years – not standardised)	0.0332 **** (0.0301 – 0.0363)	0.0334 *** (0.0303 – 0.0365)	0.0334 **** (0.0303 – 0.0365)	0.0335 **** (0.0304 – 0.0366)
Overweight/obesity norm	-0.0376 *** (-0.0575 - -0.0178)	-0.0379 *** (-0.0558 - -0.0180)	-0.0347 ** (-0.0582 to -0.0111)	-0.0344 ** (-0.0579 to -0.0108)
Overweight/obesity norm x time	0.0080 *** (0.0049 – 0.0110)	0.0080 **** (0.0049 – 0.0111)	0.0080 **** (0.0049 – 0.0111)	0.0075 **** (0.0045 – 0.0106)
Healthful food resources	-	0.0114 (-0.0082 – 0.0310)	0.0112 (-0.0084 – 0.0308)	0.0121 (-0.0075 – 0.0317)
Healthful food resources x time	-	-0.0024 (-0.0055 – 0.0007)	-0.0024 (-0.0055 – 0.0007)	-0.0027 (-0.0058 – 0.0005)
Area-level education (% university degree)	-	-	0.0062 (-0.0182 – 0.0306)	0.0040 (-0.0206 – 0.0286)
Overweight/obesity norm x healthful food resources	-	-	-	0.0208 (-0.0002 – 0.0418)
Overweight/obesity norm x healthful food resources x time	-	-	-	-0.0057 ** (-0.0092 to -0.0022)
AIC	4286.0	4287.2	4288.9	4281.6
BIC	4327.5	4333.9	4338.2	4336.1
Adjusted $ICC_{(participants)}$	0.60	0.60	0.60	0.60
Adjusted $ICC_{(State\ Suburb)}$	0.01	0.01	0.01	0.01

All models adjusted for individual covariates: age (at baseline), sex, marital status, education, employment status, and smoking status; standardised (β) coefficients and 95% confidence intervals (CI); *p < 0.05; **p < 0.01; ***p < 0.001; ****p < 0.0001; *n*=1890

Table 8.4 Associations between local descriptive insufficient fruit intake norms, food resource availability, and 10-year change in HbA_{1c}

	Model 1	Model 2	Model 3	Model 4
Predictors	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**
Insufficient fruit intake norm and fast-food outlet availability				
Time (in years – not standardised)	0.0337 **** (0.0307 – 0.0367)	0.0337 **** (0.0306 – 0.0367)	0.0337 **** (0.0307 – 0.0368)	0.0335 **** (0.0304 – 0.0365)
Insufficient fruit intake norm	0.0008 (-0.0189 – 0.0205)	0.0002 (-0.0197 – 0.0200)	0.0106 (-0.0104 – 0.0315)	0.0111 (-0.0097 – 0.0320)
Insufficient fruit intake norm*time	0.0055 *** (0.0025 – 0.0085)	0.0055 *** (0.0025 – 0.0086)	0.0055 *** (0.0025 – 0.0086)	0.0055 *** (0.0025 – 0.0086)
Fast-food outlets	-	-0.0053 (-0.0252 – 0.0146)	-0.0095 (-0.0293 – 0.0103)	-0.0096 (-0.0293 – 0.0101)
Fast-food outlets*time	-	0.0001 (-0.0030 – 0.0033)	0.0001 (-0.0030 – 0.0033)	0.0001 (-0.0031 – 0.0033)
Area-level education (% university degree)	-	-	0.0296 ** (0.0077 – 0.0514)	0.0306 ** (0.0089 – 0.0524)
Insufficient fruit intake norm*fast-food outlets	-	-	-	-0.0062 (-0.0271 – 0.0147)
Insufficient fruit intake norm*fast-food outlets*time	-	-	-	-0.0020 (-0.0054 – 0.0015)
AIC	4420.8	4424.5	4419.7	4421.4
BIC	4462.3	4471.2	4469.0	4475.9
Adjusted $ICC_{(participants)}$	0.59	0.59	0.59	0.60
Adjusted $ICC_{(State\ Suburb)}$	0.01	0.01	0.01	0.01
Insufficient fruit intake norm and healthful food resource availability				
Time (in years – not standardised)	0.0337 **** (0.0307 – 0.0367)	0.0338 **** (0.0307 – 0.0368)	0.0338 **** (0.0307 – 0.0369)	0.0337 **** (0.0306 – 0.0367)
Insufficient fruit intake norm	0.0008 *** (-0.0189 – 0.0205)	0.0020 (-0.0178 – 0.0218)	0.0123 (-0.0087 – 0.0334)	0.0115 (-0.0096 – 0.0326)
Insufficient fruit intake norm*time	0.0055 (0.0025 – 0.0085)	0.0053 *** (0.0023 – 0.0084)	0.0053 *** (0.0023 – 0.0084)	0.0052 ** (0.0021 – 0.0082)
Healthful food resources	-	0.0104 (-0.0093 – 0.0302)	0.0107 (-0.0087 – 0.0300)	0.0078 (-0.0121 – 0.0278)
Healthful food resources*time	-	-0.0015 (-0.0046 – 0.0016)	-0.0015 (-0.0049 – 0.0017)	-0.0018 (-0.0051 – 0.0015)
Area-level education (% university degree)	-	-	0.0280 ** (0.0063 – 0.0496)	0.0300 ** (0.0083 – 0.0517)
Insufficient fruit intake norm*healthful food resources	-	-	-	-0.0115 (-0.0320 – 0.0090)
Insufficient fruit intake norm*healthful food resources*time	-	-	-	-0.0011 (-0.0045 – 0.0022)
AIC	4420.8	4423.3	4419.1	4420.7
BIC	4462.3	4470.0	4468.4	4475.2
Adjusted $ICC_{(participants)}$	0.59	0.59	0.59	0.59
Adjusted $ICC_{(State\ Suburb)}$	0.01	0.01	0.01	0.01

All models adjusted for individual covariates: age (at baseline), sex, marital status, education, employment status, and smoking status; standardised (β) coefficients and 95% confidence intervals (CI); *p < 0.05; **p < 0.01; ***p < 0.001; ****p < 0.0001; *n*=1945

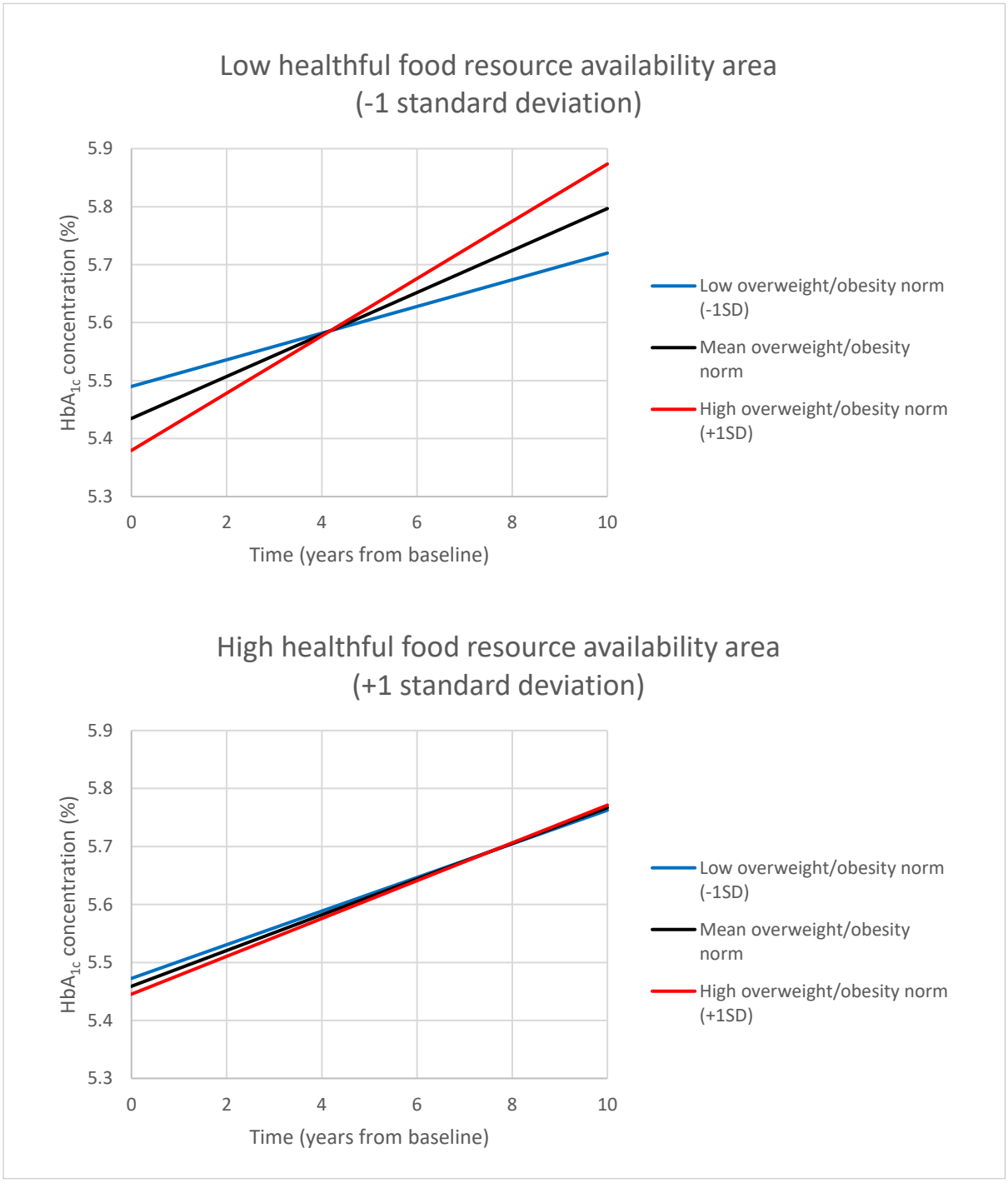

Figure 8.2 Associations between local descriptive overweight/obesity norms and HbA_{1c} trajectories according to healthful food resource availability

8.4 Discussion

Few studies have examined the influence of local descriptive health-related norms on trajectories of individual health outcomes. This study found that local descriptive norms, operationalised as the prevalence of local residents being overweight/obese or not meeting fruit intake recommendations, were each associated with the rate of increase in HbA_{1c} levels over 10 years. These relationships were robust to the inclusion of contextual measures (fast-food outlet and healthful food resource availability), area-level education, and individual-level demographic and smoking information. Fast-food outlet and healthful food resource availability were not statistically significantly associated with change in HbA_{1c} in this sample and region. However, greater healthful food resources reduced the unhealthful influence of the overweight/obesity norm on the rate of increase in HbA_{1c}. This observation supports the premise that the availability of food resources can modify relationships between local descriptive health-related norms and health outcomes.

Associations between *subjective* descriptive norms and individual health-related outcomes have previously been reported for social networks. A longitudinal (32 years) social network study found associations between norms for overweight and an individual becoming overweight [45]. Similarly, the dietary norms of peers are related to individuals' diet and dietary intentions [46,47]. The influence of geographically defined (i.e. *local*) descriptive norms on individual cardiometabolic risk has rarely been evaluated. One study found that the odds of a Dutch adult becoming overweight/obese over 13-years of follow-up increased with greater prevalence of neighbourhood overweight/obesity in models adjusted for age, sex, education and neighbourhood deprivation [16]. Similarly, an analysis using the same cohort as reported on here, documented associations between greater local descriptive overweight/obesity and physical inactivity norms, and increasing HbA_{1c} over time in models adjusted for walkability, availability of public open space, area-level education and individual-level covariates [17]. No study has thus far reported the influence of local descriptive dietary norms on individual health outcomes. The current study's findings, along with those of the two referenced studies, support the notion that local descriptive norms influence individual-level health outcomes. However, more research in different regions and populations is needed to replicate these results.

Behavioural theory suggests we imitate the behaviours of others, whether informed by direct viewing or by other informational sources [48,49]. It may be that by observing locality-based body weight (i.e., the local descriptive body weight norm) an individual determines what they consider to be a socially acceptable body weight, and that the norm overrides any known health consequences associated with a larger body size. Therefore, exposure to greater prevalence of overweight or obese persons may reduce motivation to follow health recommendations relating to diet and body weight. Interestingly, the associations between insufficient fruit intake norm and change in HbA_{1c} were similar to those for the overweight/obesity norm. The fruit intake of other residents is unlikely to be easily observed, unlike local body weight norms. As such, it is difficult to understand how the eating behaviour of nearby residents may influence individuals. Norms for overweight/obesity and insufficient fruit intake were moderately correlated (rho = 0.37, $p < 0.0001$) which may partly explain these findings. It is also possible that the similarities in results reflect broader influences such as the formation of geographically defined collective lifestyles, the expression of a shared way of relating and acting in a given environment [50,51].

Intervention strategies previously applied to reduce smoking behaviour could be adapted for use by initiatives to improve dietary behaviour. Smoking intervention has successfully changed attitudes to smoking, pushing the norm towards non-smoking due to policy intervention strategies such as increased pricing, reduced availability and limitations as to where one can smoke [52]. Similar manipulation of the food environment may assist in changing norms relating to diet behaviour and weight, particularly where norms are most unhealthful. Moreover, psychology research has shown that information on the eating behaviours of others can influence both the food selection and quantity of food consumed [53]. As such, descriptive norms information could be used to encourage increased fruit and vegetable intake [54].

This study found no association between fast-food outlet or healthful food resource availability and change in HbA_{1c}. Findings from previous studies indicate mixed results in this regard [55]. Some studies report greater fast-food outlet availability as associated with: greater weight status [56,57]; an increase in systolic and diastolic blood pressure over 1 year in low walkability neighbourhoods [58]; and mortality and hospital admissions for acute coronary syndromes [59] in models adjusted for individual and area-level covariates. Other

studies of fast-food outlet availability have reported no significant association with weight status [31,60,61]. Still other research suggests that relationships between fast-food availability and fast-food consumption [62] and cardiometabolic risk [63] are complex, being moderated by individual psychological dispositions. Similarly, when regarding healthful food resources, some studies have found associations between healthful food availability and lower 5 year diabetes incidence [64], and greater supermarket availability and reduced odds of obesity [56]. Other studies, like ours, have not observed any association between the availability of healthful food resources and cardiometabolic risk, or have observed associations in an unexpected direction [e.g., 55,61].

Food resource availability is largely viewed to function as an enabler (or conversely, a barrier, where unavailable) to obtaining and consuming desired foods. Whilst different local areas within our study region are likely to have different availabilities of food resources, all might nevertheless provide access sufficient as not to unduly limit individual dietary choices or a capacity to obtain desired foods. The median number of fast-food outlets in a buffer was five (IQR 3 – 8), suggesting that fast food was readily available across the region and lack of access would not generally be a barrier to obtaining fast food, if desired.

Though food resource availability was not associated with change in HbA_{1c} over time, healthful food resource availability modified the association between the overweight/obesity norm and change in HbA_{1c} over time. In areas with a greater availability of healthful food resources, the impact of a greater overweight/obesity norm on rising HbA_{1c} was reduced. Conversely, where there was a lesser availability of healthful food resources, the rate of increase in HbA_{1c} due to a greater overweight/obesity norm was amplified. Interactions between environmental features in relation to cardiometabolic risk have rarely been studied [5]. No previous studies have reported on the presence or absence of interactions between local descriptive health-related norms and the contextual food environment in relation to HbA_{1c} or other health outcomes. Further study of the interactive, joint effects of contextual and compositional risk conditions on chronic disease outcomes is required to inform strategies for intervention design and targeting. Local environments that predispose *and* enable individual health behaviours will be more supportive of health than environments that support only one or the other set of factors. Attention to local health-related norms (to predispose healthful

behaviour) together with the provision of sufficient resources (to enable healthful behaviour) is needed to reduce chronic disease outcomes [65,66].

It is necessary also to develop an understanding of how local descriptive norms are shaped. Intervention strategies intending to change local norms will need to be assessed for effectiveness. Appropriate intervention strategies may need to differ according to the level of the current descriptive norm. Applying Rogers' Diffusion of Innovations theory [67], where the local prevalence of a positive behavioural norm is low, intervention strategies might prioritise the targeting of early adopters. Conversely, where the local prevalence of a positive behavioural norm is moderate, targeting laggards may be more appropriate. Strategies might further differ depending on the target group. For example, strategies aimed at early adopters could involve health education campaigns that appeal to values, attitudes and beliefs which predispose behaviour, while those targeting the early majority could focus on enabling mechanisms such as the provision of healthful food at affordable cost. Further research is necessary to empirically measure and to implement interventions to shape and apply healthful norms.

8.4.1 Strengths and limitations

Strengths of this study include the use of a 10-year population-based cohort with three waves of data including clinical measures. The longitudinal design supports causal inference through temporality of measures [2]. However, the positive longitudinal associations between the local descriptive overweight/obesity norm and increasing HbA_{1c} differed from the cross-sectional results which indicated an inverse association between the overweight/obesity norm and baseline HbA_{1c}. Sub-analyses indicated that these unexpected cross-sectional findings were carried primarily by older participants and that age adjustment did not fully remove the impact of these influences. Emphasis should be on the longitudinal findings as the cross-sectional results are likely spurious.

The outcome (HbA_{1c}) was clinically measured, avoiding self-report bias. However, individual demographic and smoking information were self-reported, and the local descriptive norms were aggregated from self-report survey data with the consequent possibility of self-report bias.

The methods and data sources used to operationalise the environmental exposures were strengths of this study. The contextual food environment was represented using objective measures extracted from a database constructed from data collected by field surveyors [37]. Local descriptive norms data were derived from a separate survey, thus avoiding same-sample bias [68]. Environmental exposures were defined using ego-centred road-network buffers, as has been previously recommended [69], with local-area education expressed using a spatial unit designed to closely match with these road-network buffers. The use of differently sized road-network buffers would have added to this research, however this was not possible, as previously outlined. It is also important to note the possibility that self-selection into neighbourhoods may have influenced this study's results. This, however, is of greater concern in cross-sectional studies than those with a longitudinal design [70].

Lastly, a basic premise of this study is that people are influenced by their *local* residential environments and their opportunities to access resources within these areas. This does not account for time spent proximal to their place of residence, and opportunities and exposures provided within the work-place and other destinations, or while commuting. The influence of local residential exposures may vary according to time spent in the local residential area, which may itself relate to individual-level sociodemographic factors and lifestyle choices. Older individuals, or those caring for young children at home, may spend more time close to home and thus be more strongly influenced by local environment exposures. Future research will use technologies such as GPS tracking to assess time spent within different geographies for individuals. Consideration of transport modes may also be important. Car ownership may modify relationships between residential exposures and health outcomes. In the current study region, cars are the predominant mode of transport though public transport options are available and streets are generally walkable with adequate footpaths provided. These may be important factors to consider in future studies.

8.5 Conclusion

Local descriptive body weight and dietary norms reflect compositional population characteristics. Food resource availability reflects context. The assessment of compositional norms in relation to health outcomes has rarely been investigated. This longitudinal study found only compositional norms, not food resource availability, to be associated with 10-year change in HbA_{1c}. However, the availability of healthful food resources modified the relationship between the local descriptive overweight/obesity norm and rate of change in HbA_{1c}.

Research in different populations and regions is recommended to replicate these results. It is also recommended that future research investigate how compositional norms may be shaped, and the mechanisms through which compositional norms influence individual health outcomes. The findings of this study suggest that compositional norms should be considered in intervention strategies targeting cardiometabolic risk.

Abbreviations

AIC: Akaike information criterion; BIC: Bayesian information criterion; BMI: body mass index; CD: Census Collection District; CI: confidence interval; CVD: cardiovascular disease; HbA_{1c}: glycosylated haemoglobin; ICC: intraclass correlations; IQR: interquartile range; NWAHS: North West Adelaide Health Study; PAMS Project: Place and Metabolic Syndrome Project; SAMSS: South Australian Monitoring and Surveillance System; SD: standard deviation; SES: socioeconomic status

Declarations

Ethics approval and consent to participate

This study was part of the Place and Metabolic Syndrome (PAMS) Project which aimed to assess the influence of social and built environmental factors on the evolution of cardiometabolic risk. The PAMS Project received ethical approval from the University of South Australia, Central Northern Adelaide Health Service, Queen Elizabeth Hospital, and South Australian Department for Health and Ageing Human Research Ethics Committees. Written informed consent of NWAHS cohort participants was obtained prior to each wave of data collection.

Consent for publication

The research presented in this manuscript is not a case study, nor does not contain any individual person's data in any form. This section is, therefore, not applicable.

Availability of data and materials

The ethics approvals granted for this research do not include consent for the sharing of the datasets supporting the conclusions of this article. The spatial nature of the data provides a risk to participant identifiability and confidentiality. Further, the provider of data from the longitudinal cohort, a branch of the state government, has not agreed to these data being made publicly available.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

SJC, MD and CP conceived and designed the study. SJC analysed the data with input from CP and TN. SJC, MD, CP, NJH and NTC contributed to interpretation of results. SJC wrote the manuscript, and MD, CP, NJH, NTC, TN, AWT and RJA revised it critically for important intellectual content. All authors approved the final manuscript.

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References

1. Daniel M, Lekkas P, Cargo M, Stankov I, Brown A: **Environmental risk conditions and pathways to cardiometabolic diseases in Indigenous populations.** *Annu Rev Publ Health* 2011, **32:**327-347.
2. Daniel M, Moore S, Kestens Y: **Framing the biosocial pathways underlying associations between place and cardiometabolic disease.** *Health & Place* 2008, **14:**117-132.
3. Black C, Moon G, Baird J: **Dietary inequalities: what is the evidence for the effect of the neighbourhood food environment?** *Health & Place* 2014, **27:**229-242.
4. Macintyre S, Ellaway A, Cummins S: **Place effects on health: how can we conceptualise, operationalise and measure them?** *Soc Sci Med* 2002, **55:**125-139.
5. Leal C, Chaix B: **The influence of geographic life environments on cardiometabolic risk factors: a systematic review, a methodological assessment and a research agenda.** *Obes Rev* 2011, **12:**217-230.
6. Mobley LR, Root ED, Finkelstein EA, Khavjou O, Farris RP, Will JC: **Environment, obesity, and cardiovascular disease risk in low-income women.** *Am J Prev Med* 2006, **30:**327-332.
7. Fraser LK, Edwards KL, Tominitz M, Clarke GP, Hill AJ: **Food outlet availability, deprivation and obesity in a multi-ethnic sample of pregnant women in Bradford, UK.** *Soc Sci Med* 2012, **75:**1048-1056.
8. Ajzen I: **The theory of planned behavior.** *Organ Behav Hum Dec* 1991, **50:**179-211.
9. Cialdini RB, Reno RR, Kallgren CA: **A focus theory of normative conduct: recycling the concept of norms to reduce littering in public places.** *J Pers Soc Psychol* 1990, **58:**1015-1026.
10. Deutsch M, Gerard HB: **A study of normative and informational social influences upon individual judgment.** *J Abnorm Soc Psych* 1955, **51:**629-636.
11. Carrus G, Bonnes M, Fornara F, Passafaro P, Tronu G: **Planned behavior and "local" norms: an analysis of the space-based aspects of normative ecological behavior.** *Cogn Process* 2009, **10:**198-200.
12. Fornara F, Carrus G, Passafaro P, Bonnes M: **Distinguishing the sources of normative influence on proenvironmental behaviors: the role of local norms in household waste recycling.** *Group Processes & Intergroup Relations* 2011, **14:**623-635.
13. Kormos C, Gifford R, Brown E: **The influence of descriptive social norm information on sustainable transportation behavior: a field experiment.** *Environ Behav* 2015, **47:**479-501.
14. Nigbur D, Lyons E, Uzzell D: **Attitudes, norms, identity and environmental behaviour: using an expanded theory of planned behaviour to predict participation in a kerbside recycling programme.** *Brit J Soc Psychol* 2010, **49:**259-284.
15. Christakis NA, Fowler JH: **Social contagion theory: examining dynamic social networks and human behavior.** *Stat Med* 2013, **32:**556-577.
16. Blok DJ, de Vlas SJ, van Empelen P, Richardus JH, van Lenthe FJ: **Changes in smoking, sports participation and overweight: does neighborhood prevalence matter?** *Health & Place* 2013, **23:**33-38.
17. Carroll SJ, Paquet C, Howard NJ, Coffee NT, Taylor AW, Niyonsenga T, Daniel M: **Local descriptive norms for overweight/obesity and physical inactivity, features of the built environment, and 10-year change in glycosylated haemoglobin in an Australian population-based biomedical cohort.** *Social Science & Medicine* 2016, **166:**233-243.
18. ABS: **Usual Residents Profile 2001, cat. no. 2004.0 [Online].** vol. 2011. Canberra: Australian Bureau of Statistics; 2003.
19. ABS: **Statistical Geography Volume 2: Census Geographic Areas Australia.** Canberra: Australian Bureau of Statistics; 2001.
20. South Australian Department of Health: **HOS: Self Reported Prevalence of Obesity in the SA Health Regions.** Adelaide: Population Research and Outcome Studies Unit, South Australian Department of Health; 2005.

21. Dal Grande E, Taylor A, Hurst B, Kenny B, Catcheside B: **The Health Status of People Living in the South Australian Divisions of General Practice: South Australian Monitoring and Surveillance System July 2002 - December 2003.** Adelaide: Population Research and Outcome Studies Unit, South Australian Department of Health; 2004.
22. Wilcox S, Castro C, King AC, Housemann R, Brownson RC: **Determinants of leisure time physical activity in rural compared with urban older and ethnically diverse women in the United States.** *J Epidemiol Commun H* 2000, **54:**667-672.
23. Grant J, Chittleborough C, Taylor A, Dal Grande E, Wilson D, Phillips P, Adams R, Cheek J, Price K, Gill T, Ruffin R: **The North West Adelaide Health Study: detailed methods and baseline segmentation of a cohort for chronic diseases.** *Epidemiological Perspectives and Innovations* 2006, **3**.
24. ABS: **Census of Population and Housing: CDATA 2001 Datapack - Usual Residents Profile, 2001** (Statistics ABo ed. Canberra, Australia: Australian Bureau of Statistics; 2001.
25. Grant J, Taylor A, Ruffin R, Wilson D, Phillips P, Adams R, Price K: **Cohort profile: The North West Adelaide Health Study (NWAHS).** *Int J Epidemiol* 2009, **38:**1479-1486.
26. Bennett CM, Guo M, Dharmage SC: **HbA1c as a screening tool for detection of Type 2 diabetes: a systematic review.** *Diabetic Med* 2007, **24:**333-343.
27. IEC: **International Expert Committee Report on the role of the A1C assay in the diagnosis of diabetes.** *Diabetes Care* 2009, **32:**1327-1334.
28. Khaw K-T, Wareham N, Bingham S, Luben R, Welch A, Day N: **Association of hemoglobin A1c with cardiovascular disease and mortality in adults: the European Prospective Investigation into Cancer in Norfolk.** *Ann Intern Med* 2004, **141:**413-420.
29. Bohannon RW: **Comfortable and maximum walking speed of adults aged 20—79 years: reference values and determinants.** *Age and Ageing* 1997, **26:**15-19.
30. Astell-Burt T, Feng X: **Geographic inequity in healthy food environment and type 2 diabetes.** *MJA* 2015, **203**.
31. Jeffery RW, Baxter J, McGuire M, Linde J: **Are fast food restaurants an environmental risk factor for obesity?** *Int J Behav Nutr Phy* 2006, **3**.
32. Reitzel LR, Regan SD, Nguyen N, Cromley EK, Strong LL, Wetter DW, McNeill LH: **Density and proximity of fast food restaurants and body mass index among African Americans.** *Am J Public Health* 2014, **104:**110-116.
33. National Health and Medical Research Council: **Australian Dietary Guidelines.** Canberra: National Health and Medical Research Council; 2013.
34. WHO: **Global Database on Body Mass Index: An Interactive Surveillance Tool for Monitoring Nutrition Transition** [http://apps.who.int/bmi/index.jsp]
35. Population Research and Outcome Studies: **South Australian Monitoring and Surveillance System: Survey Methodology** [http://health.adelaide.edu.au/pros/docs/reports/report_samss_tech_paper.pdf]
36. Population Research and Outcome Studies: **South Australian Monitoring and Surveillance System (SAMSS)** [http://health.adelaide.edu.au/pros/data/samss/]
37. South Australian Government: **South Australian Retail Database.** Adelaide, South Australia: Department of Planning, Transport and Infrastructure (DPTI); 2007.
38. Spatial Planning Analysis and Research Unit: *Guide to using the 2007 Retail Database Adelaide Statistical Division and nearby large Country Centres.* Adelaide: Planning SA; 2008.
39. Turrell G, Giskes K: **Socioeconomic disadvantage and the purchase of takeaway food: a multilevel analysis.** *Appetite* 2008, **51:**69-91.
40. Murray GD, Findlay JG: **Correcting for the bias caused by drop-outs in hypertension trials.** *Stat Med* 1988, **7:**941-946.
41. ABS: **Basic Community Profile (BCP) DataPack, cat. no. 20069.0.30.001 (Second Release).** Canberra: Australian Bureau of Statistics; 2006.
42. ABS: **Australian Standard Geographic Classification Vol. 2: Census Geographic Areas, Australia 2006.** Canberra: Australian Bureau of Statistics; 2006.

43. West BT, Welch KB, Galecki AT: *Linear Mixed Models: A Practical Guide Using Statistical Software.* Boca Raton: Chapman & Hall/CRC; 2007.
44. Ukoumunne O, Gulliford M, Chinn S, Sterne J, Burney P: **Methods for evaluating area-wide and organisation-based interventions in health and health care: a systematic review.** *Health Technol Asses* 1999, **3:**98.
45. Christakis NA, Fowler JH: **The spread of obesity in a large social network over 32 years.** *New Engl J Med* 2007, **357:**370-379.
46. Pachucki MA, Jacques PF, Christakis NA: **Social network concordance in food choice among spouses, friends, and siblings.** *Am J Public Health* 2011, **101:**2170-2177.
47. Tuu HH, Olsen SO, Thao DT, Anh NTK: **The role of norms in explaining attitudes, intention and consumption of a common food (fish) in Vietnam.** *Appetite* 2008, **51:**546-551.
48. Rivis A, Sheeran P: **Descriptive norms as an additional predictor in the theory of planned behaviour: a meta-analysis.** *Curr Psychol* 2003, **22:**218-233.
49. Bandura A: *Social Learning Theory.* New York City: General Learning Press; 1971.
50. Frohlich KL, Corin E, Potvin L: **A theoretical proposal for the relationship between context and disease.** *Sociol Health Ill* 2001, **23:**776-797.
51. Bernard P, Charafeddine R, Frohlich KL, Daniel M, Kestens Y, Potvin L: **Health inequalities and place: a theoretical conception of neighbourhood.** *Soc Sci Med* 2007, **65:**1839-1852.
52. Ashe M, Graff S, Spector C: **Changing places: policies to make a healthy choice the easy choice.** *Public Health* 2011, **125:**889-895.
53. Robinson E, Thomas J, Aveyard P, Higgs S: **What everyone else is eating: a systematic review and meta-analysis of the effect of informational eating norms on eating behavior.** *J Acad Nutr Diet* 2014, **114:**414-429.
54. Robinson E, Fleming A, Higgs S: **Prompting healthier eating: testing the use of health and social norm based messages.** *Health Psychol* 2014, **33:**1057-1064.
55. Daniel M, Paquet C, Auger N, Zang G, Kestens Y: **Association of fast-food restaurant and fruit and vegetable store densities with cardiovascular mortality in a metropolitan population.** *Eur J Epidemiol* 2010, **25:**711-719.
56. Bodor JN, Rice JC, Farley TA, Swalm CM, Rose D: **The association between obesity and urban food environments.** *J Urban Health* 2010, **87:**771-781.
57. Mehta NK, Chang VW: **Weight status and restaurant availability: a multilevel analysis.** *Am J Prev Med* 2008, **34:**127-133.
58. Li F, Harmer P, Cardinal BJ, Vongjaturapat N: **Built environment and changes in blood pressure in middle aged and older adults.** *Prev Med* 2009, **48:**237-241.
59. Alter DA, Eny K: **The relationship between the supply of fast-food chains and cardiovascular outcomes.** *Can J Public Health* 2005, **96:**173-177.
60. Simmons D, McKenzie A, Eaton S, Cox N, Khan MA, Shaw J, Zimmet P: **Choice and availability of takeaway and restaurant food is not related to the prevalence of adult obesity in rural communities in Australia.** *Int J Obesity* 2005, **29:**703-710.
61. Wang MC, Kim S, Gonzalez AA, MacLeod KE, Winkleby MA: **Socioeconomic and food-related physical characteristics of the neighbourhood environment are associated with body mass index.** *J Epidemiol Commun H* 2007, **61:**491-498.
62. Paquet C, Daniel M, Knäuper B, Gauvin L, Kestens Y, Dubé L: **Interactive effects of reward sensitivity and residential fast-food restaurant exposure on fast-food consumption.** *Am J Clin Nutr* 2010, **91:**771-776.
63. Paquet C, Dube L, Gauvin L, Kestens Y, Daniel M: **Sense of mastery and metabolic risk: moderating role of the local fast-food environment.** *Psychosocial Medicine* 2010, **72:**324-331.
64. Auchincloss AH, Diez Roux AV, Mujahid MS, Shen M, Bertoni A, Carnethon M: **Neighborhood resources for physical activity and healthy foods and incidence of type 2 diabetes mellitus: the multi-ethnic study of Atherosclerosis.** *Arch Intern Med* 2009, **169:**1698-1704.
65. Green LW, Richard L, Potvin L: **Ecological foundations of health promotion.** *Am J Health Promot* 1996, **10:**270-281.

66. Daniel M, Green LW: **Health promotion and education.** In *Encyclopedia of Public Health. Volume* 2. Edited by Breslow L. New York: Macmillan; 2002: 541-548
67. Rogers EM: *Diffusion of Innovations.* Simon and Schuster; 2010.
68. Diez Roux AV: **Neighborhoods and health: where are we and where do we go from here?** *Revue d'Epidémiologie et de Santé Publique* 2007, **55:**13-21.
69. Chaix B, Merlo J, Evans D, Leal C, Havard S: **Neighbourhoods in eco-epidemiologic research: delimiting personal exposure areas. A response to Riva, Gauvin, Apparicio and Brodeur.** *Soc Sci Med* 2009, **69:**1306-1310.
70. Boone-Heinonen J, Gordon-Larsen P, Guilkey DK, Jacobs Jr DR, Popkin BM: **Environment and physical activity dynamics: the role of residential self-selection.** *Psychol Sport Exerc* 2011, **12:**54-60.

Chapter 9

Discussion

9 Discussion

This research is founded on the notion that slowing the rising levels of cardiometabolic disease requires understanding the independent and interactive contributions of environmental features, compositional and contextual, to cardiometabolic disease. Though review articles have highlighted the need for research to evaluate the interactive contributions on environmental features on cardiometabolic risk outcomes (e.g., Leal & Chaix 2011; Lovasi, Grady & Rundle 2012; Schüle & Bolte 2015), few studies have done so. In addition, one group of compositional factors, local descriptive norms, has rarely been assessed in relation to cardiometabolic health outcomes. No studies have assessed the interactive effects of local descriptive norms and built environment features on change in cardiometabolic risk. This dissertation attends to these research gaps.

The aim of this dissertation was to advance understanding of the relationships between environmental risk conditions and health outcomes by assessing the independent and interactive contributions of local descriptive norms and built environment features to 10-year change in individual-level cardiometabolic risk. Three research objectives derived from this aim, one to assess the utility of a composite index, the others extending to four testable hypotheses (refer to Table 4.1).

Chapters 6, 7 and 8 presented the research addressing the objectives and hypotheses. This chapter relates the study results back to the dissertation objectives and hypotheses, and interprets the findings in the broader context of research evaluating relationships between environmental risk conditions and health outcomes.

This discussion chapter consists of six main sections. The first two sections (**Section 9.1** Objective 1 and **Section 9.2** Objectives 2 and 3) briefly summarise the findings from the research studies. These two sections do not provide detailed discussion of these findings, but highlight how the included studies and their findings relate to the research hypotheses. Implications for policy and public health practice are presented in **Section 9.3**. **Section 9.4** describes the strengths and limitations of the overall research. **Section 9.5** discusses the implications for advancing the research field, and **Section 9.6** concludes the dissertation.

9.1 Objective 1

This objective was to validate a continuous index of cardiometabolic risk constructed from five clinical risk factors, and assess its utility in relation to change in cardiometabolic risk over time. The rationale was a perceived need to use the cCICR as the outcome measure for Studies 2 and 3 to express change in cardiometabolic risk over time. The majority of studies assessing associations between environmental features and cardiometabolic risk have used a single cardiometabolic risk factor, such as obesity, as the outcome. This is despite recommendations that would preference composite measures over the use of single measures of cardiometabolic risk (Jackson et al. 2005; Perk et al. 2012).

Commonly used composite cardiometabolic risk indices often include both clinical (e.g., blood glucose levels) and non-clinical (e.g., health behaviour, family history, and area-level SES) information (e.g., D'Agostino et al. 2008; Hippisley-Cox et al. 2007; Wilson et al. 2007; Woodward, Brindle & Tunstall-Pedoe 2007), thus limiting their utility in inferential research where non-clinical factors may be of interest as predictors and therefore cannot be part of the outcome measure. cCICRs provide a global expression of cardiometabolic risk, objectively constructed solely from clinical measures and expressed in a continuous format to provide information on risk severity. Using a continuous outcome measure, as compared to a categorical measure, can increase model sensitivity and analysis power (Ragland 1992). Constructing the cCICR solely from clinical factors can remove self-report bias. Moreover, using only clinical factors to construct the cCICR can enable behavioural and environmental information to be included as predictors within analysis models that use the risk index as an outcome.

Two cCICRs were constructed: 1) a cCICR-Z as a sum of z-scores; and 2) a cCICR-PCA as the weighted sum of the first two principal components from PCA with the proportion of variance explained used as weights. In the initial validation study, the two cCICRs performed similarly well to the Framingham Risk Scores for CVD and Type 2 Diabetes (D'Agostino et al. 2008; Wilson et al. 2007), with no statistically significant differences in predictive accuracies between the risk indices.

The initial validation of the cCICRs, however, did not consider time, and further assessment of the cCICR-Z was conducted to evaluate its performance in measuring *change* in

cardiometabolic risk over time. The cCICR-Z was selected over the cCICR-PCA as its construction was simpler than that for the cCICR-PCA. Multilevel models were used to estimate how time (in years) predicted the cCICR-Z, its five component risk factors and HbA_{1c}, expressed at three time points. The cCICR-Z did not behave as expected over time, and differential responses for the component cardiometabolic risk factors were observed. Though three cCICR component factors worsened over time, namely waist circumference, and blood triglyceride and glucose concentrations, the remaining two risk factors, MAP and HDL improved. These findings support the premise that cardiometabolic risk factors may respond differentially to environmental and behavioural exposures (Boulé et al. 2001; Dhingra et al. 2007; Paquet et al. 2014) though this premise was not explicitly tested and is not the focus of this dissertation.

While cCICRs have previously been used to assess change in cardiometabolic risk within studies of shorter time frames, including change in cardiometabolic risk in response to exercise interventions (e.g., Bateman et al. 2011; Johnson et al. 2007), the utility of such an index in longitudinal research involving biomedical cohorts has yet to be established. While further consideration of the utility of cCICRs in longitudinal research is recommended, research focusing on the differential responses of risk factors to environmental and behavioural exposures might optimally take priority. Such research would lead to better understanding of the specific pathways of influence from environmental risk conditions to cardiometabolic outcomes and consequently could be used to inform intervention design. Moreover, use of composite indices in longitudinal research may disguise actual associations between environmental influences and cardiometabolic outcomes. Null findings in papers that have used a composite index may be due to differential changes in component risk factors rather than a lack of association between environmental features and cardiometabolic outcomes.

HbA_{1c} was considered as an alternative to the cCICR-Z in expressing change in cardiometabolic risk for Studies 2 and 3 due to its strong linear association with cardiometabolic disease (ADA 2015; Eeg-Olofsson et al. 2010; Khaw et al. 2004; Nathan et al. 2013). Use of HbA_{1c} allowed for the investigation of a risk factor specific response to environmental exposures while providing a continuous expression of risk. In the analyses assessing change over time, HbA_{1c} behaved as expected (i.e., increased as the cohort aged). HbA_{1c} is associated with both CVD

and type 2 diabetes, independently of other risk factors (Eeg-Olofsson et al. 2010; Khaw et al. 2004; Nathan et al. 2013).

Consideration of appropriate expressions of cardiometabolic outcomes within empirical research is important for methodological rigour. This dissertation adds to knowledge on the expression of cardiometabolic risk and identifies additional research areas (Section 9.5.1, page 224) needed to further develop rigour in the expression of cardiometabolic outcomes. The overall (and unanticipated) findings from Study 1, attending Objective 1, informed the selection of the measure used to represent cardiometabolic risk (HbA_{1c}) in Studies 2 and 3.

9.2 Objectives 2 and 3

Objective 2 was to evaluate the independent contributions of compositional and contextual features to change in cardiometabolic risk over 10 years. From this derived an hypothesis about composition, Hypothesis 1, specifically: *greater level of adverse local descriptive norm (overweight/obesity, physical inactivity, and insufficient fruit intake) would predict increases in cardiometabolic risk over time*. Two hypotheses pertaining to context also derived from Objective 2: Hypothesis 2*, greater level of positive feature (walkability, POS availability and healthful food availability) would predict lesser increases in cardiometabolic risk over time*; and Hypothesis 3, *greater level of adverse feature (fast-food availability) would predict increasing cardiometabolic risk over time*.

Objective 3 was to evaluate the joint effects of compositional and contextual features to change in cardiometabolic risk. From this derived Hypothesis 4, having three parts. Hypothesis 4a pertained to the interaction of adverse composition and adverse context, specifically: *greater level of adverse local descriptive norm (e.g., overweight/obesity) and greater level of adverse feature (e.g., fast-food availability) would interact to amplify rising cardiometabolic risk over time*. Hypothesis 4b concerned the interaction of favourable composition x favourable context: *lesser level of adverse local descriptive norm (e.g., overweight/obesity) and greater level of positive feature (e.g., walkability) would interact to attenuate rising cardiometabolic risk over time*. Hypothesis 4c pertained to the interaction of favourable composition x adverse context, specifically: *lesser level of adverse local descriptive norm (e.g., physical inactivity) would interact with level of adverse feature (e.g., lesser walkability) to attenuate rising cardiometabolic risk over time*.

Studies 2 and 3 found local descriptive norms for overweight/obesity, physical inactivity, and insufficient fruit intake were each associated with worsening trajectories of cardiometabolic risk (expressed as HbA_{1c}) over 10 years, in models accounting for individual-level demographic and smoking information. These relationships were robust to the inclusion of built environment features: walkability; POS availability; and fast-food and healthful food outlet availability; as well as area-level education (included as a covariate). Importantly, these findings support Hypothesis 1, providing evidence that local descriptive norms can influence individual-level health outcomes and should therefore be considered as environmental risk conditions. This indicates a clear need to consider local descriptive norms within public health

practice and policy (discussed in Section 9.3). An expectation that individuals can change their behaviour when situated in an area with adverse local descriptive norms may be unrealistic, treating individuals as separate from their environments, whereas individuals interact with, influence and are influenced by their environments.

For our study region and the participants in our cohort, greater walkability was associated with a reduction in the rate of increase in cardiometabolic risk over 10 years in models including local descriptive norms, area-level education and individual-level covariates. Fast-food outlet and healthful food resource availability were not associated with change in HbA_{1c}. Contrary to expectations, greater POS availability was associated with rising HbA_{1c} concentration over time. These findings provide some support for Hypothesis 2 and add further support to the current view that the built environment influences individual-level health. Hypothesis 3 was not supported. Numerous previous studies have assessed features of the built environment hypothesised to be related to physical activity and food behaviour, though these studies have often not undertaken simultaneous analysis of different dimensions of environments to assess their independent effects (Leal & Chaix 2011). The unique contribution this dissertation provides is the analysis of compositional norms concurrently with features of the built environment in models predicting change in HbA_{1c} over time.

The findings from Studies 2 and 3 also support the premise of interactive effects between compositional and contextual features and cardiometabolic outcomes (Objective 3, Hypothesis 4). Local descriptive overweight/obesity norm interacted with walkability and healthful food availability. The magnitude of associations between local overweight/obesity norms and increasing HbA_{1c} was attenuated for contexts of greater walkability or healthful food availability and exacerbated for contexts of lesser walkability or healthful food availability. No research has previously reported on the interactions between local descriptive norms and the physical activity and food environments in relation to cardiometabolic risk. The findings from Studies 2 and 3 provide empirical support that different dimensions of environments interact in shaping chronic disease risk. Understanding which environmental features are related to health outcomes is important to design successful public health initiatives, informing as to which environmental features to best act upon. Developing this understanding further to include the conditions under which these relations are important by

investigating interaction effects is necessary to refine policy and interventions strategies. Potential strategies are presented in Section 9.3.

9.2.1 Composition beyond area-level socioeconomic status

In much of the research regarding place effects on health, context has referred to neighbourhood effects (area or group effects) independent of individual-level effects (Diez Roux 2001). This approach has typically framed as *contextual* the remaining influence of area-level SES on health outcomes after having accounted in statistical models for individual-level factors. Correspondingly, the variance accounted for by individual-level factors, primarily socioeconomic, has been considered to reflect *compositional* influences (Diez Roux 2001; Ross, CE & Mirowsky 2008). Composition, however, can defensibly be considered to be more than the impact of individual influences simply accounted for in statistical models as "noise" or nuisance factors to be removed. The explicit modelling of area-level SES, or *any* collective attribute expressed spatially, while still accounting for the contributions of individual-level SES and other individual-level covariate, can enable assessment of spatial variations in collective lifestyles. Thus, to expand understanding of how environments shape health, it is necessary to reframe and to explicitly model compositional influences as population phenomena, as distinct and separate from individual-level covariates that relate to health outcomes for the survey or cohort participants being studied.

The findings of Studies 2 and 3 indicate that local descriptive health-related norms may be important but overlooked compositional influences on individual-level health outcomes. This supports the premise that local descriptive norms, a component of collective lifestyle, represent more than the sum of individual-level attitudes and behaviours expressed only for survey or cohort participants. At the population level, local descriptive norms, reflecting the spatially variable social milieu for a given area, would seem to reflect a social pressure that consciously or subconsciously influences individuals' beliefs, behaviours and health.

Very few studies have conceived and analysed compositional factors in this way. Of those that have, a longitudinal study in the US with 9 years of follow up reported associations between decreasing residential area co-ethnic concentration over time and increasing BMI amongst Chinese immigrants aged 45-84 years (Lê-Scherban et al. 2014). A cross-sectional US study reported a greater odds of being obese for individuals residing in areas with greater

proportions of Blacks compared to areas with lesser proportions of Blacks, after accounting for area-level SES and individual-level sociodemographic factors (Boardman et al. 2005). This association became null after the inclusion of area-level obesity prevalence (i.e., local descriptive obesity norm) in analytic models, suggesting that variations in neighbourhood obesity prevalence may mediate the relationship between neighbourhood racial composition and the odds of obesity among residents of Black communities (Boardman et al. 2005).

This dissertation, and manuscripts 2 and 3 specifically, contribute to research on environmental influences on health through the focus on a novel compositional feature, local descriptive norms. The results of this work highlight the need for further conceptual, theoretical and empirical consideration of local descriptive norms. Such research also has implications for public and population health research and practice, especially intervention design.

9.2.2 Conceptualisation of norms

'Norms' feature in many sociology and psychology theories aiming to explain human behaviour (e.g., Ajzen 1991; Christakis & Fowler 2013; Cialdini, Reno & Kallgren 1990; Fishbein 2008; Gibbs 1981; Granovetter 1978; Montano & Kasprzyk 2008; Rivis & Sheeran 2003). However, 'norms' have rarely been well defined within research and theory, hindering efforts to understand the influences of 'norms' on behaviour and health. Various studies have conceived 'norms' differently. This dissertation has outlined and defined social norms based on the Focus Theory of Normative Conduct (injunctive and descriptive norms) (Cialdini, Reno & Kallgren 1990). These definitions were refined in terms of local and subjective norms (Carrus et al. 2009; Fornara et al. 2011; Kormos, Gifford & Brown 2015). Drawing on these definitions, Studies 2 and 3 of this dissertation assessed the influence of local descriptive norms on cardiometabolic risk, finding that local descriptive norms predicted worsening individual-level trajectories of HbA_{1c}. What this means for policy and public health practice is presented in the next section.

Previous studies have established the benefits in differentiating injunctive and descriptive norms (Borsari & Carey 2001; Cialdini, Reno & Kallgren 1990; Fornara et al. 2011; Rivis & Sheeran 2003) and local and subjective norms (Carrus et al. 2009; Fornara et al. 2011; Kormos, Gifford & Brown 2015). Many of these studies have focused on environmental behaviours

such as littering, recycling, and household energy efficiency choices. This dissertation broadens the application of these classifications of 'norms', introducing the set of definitions to research regarding the influence of environmental risk conditions on individual-level health outcomes. Though only local descriptive norms were analysed, the findings reported here support the use of such a definition in research assessing environmental influences on health. The implications for advancing the research field are presented in Section 9.5.

'Norms' have typically been considered within the social sphere, that is, as a social influence defined between individuals connected by strong affective ties such as family and friends (e.g., Theory of Planned Behaviour and Social Contagion Theory; Ajzen 1991; Christakis & Fowler 2013, respectively). This approach has ignored the influence imparted simply through sharing the same 'space', limiting the development of understanding these influences. This dissertation highlights the importance of considering norms outside of the social sphere, that is, local norms, in relation to individual health.

9.3 Implications for policy and public health practice

To effectively combat rising rates of cardiometabolic disease (and other chronic conditions) an integrated approach to health intervention design is needed including multi-level and multi-sectoral strategies based on scientific evidence and on-going research (WHO 2013). Such strategies will require a supportive policy and legislative framework for success (WHO 2013). This dissertation adds to the scientific evidence linking residential environments to health outcomes and introduces a group of novel environmental features, local descriptive norms, that can be used both as a focus of interventions (i.e., an environmental feature to act upon) and as a marker of residential areas at increased cardiometabolic risk. Further, the results from Study 2 support previous findings indicating the importance of walkability to health outcomes, and thus its importance to intervention design. Lastly, the findings from both Study 2 and Study 3 indicate interactive influences of environmental features in relation to cardiometabolic risk. Consequently, interventions should evaluate, and consider intervening upon, multiple environmental features simultaneously to maximise intervention effectiveness.

9.3.1 Policy and legislation

Policies can be used to promote or discourage behaviours and traits associated with chronic diseases, including cardiometabolic disease, by targeting social norms and the built environment (Ashe, Graff & Spector 2011). The built environment can be shaped by land use planning and urban design legislation and policies influencing the placement of food stores, aspects of walkability (e.g., population/housing density, street connectivity, land use mix), features of street scapes (e.g., quality paving, lighting, traffic flow and safety), and the amount and quality of POS including associated features (e.g., sporting fields and courts, playgrounds, BBQs, shelter, shade, benches, toilets). Health promotion strategies can support such policies by educating the public (e.g., media campaigns) as to the benefits of such built environment changes. Moreover, population wide education may motivate local advocacy in areas where needed built environment changes are absent or slow. In addition, traditional education strategies can be used to motivate individuals to change their behaviours and take advantage of more health supportive built environments.

9.3.1.1 Denormalisation strategies

Legislation and policy may shift population norms by creating daily environments where the healthier choice is the easier choice (Ashe, Graff & Spector 2011). For example social and industry denormalisation (Hammond, D et al. 2006; Roeseler & Burns 2010) strategies and choice architecture (Thaler, Sunstein & Balz 2013) can be used to shift default choices in a more healthful direction. Social denormalisation strategies aim to "change the broad social norms" shifting a behaviour from being a "normal, desirable practice to being an abnormal practice" (Tobacco Control Section 1998, p 3). Industry denormalisation supports social denormalisation by exposing and counteracting industry practices such as manipulative media and marketing strategies (Hammond, D et al. 2006; Hershey et al. 2005; Roeseler & Burns 2010). Social and industry denormalisation strategies have created a social and legal environment wherein smoking became "less desirable, less acceptable, and less accessible", successfully reducing smoking prevalence (Ashe, Graff & Spector 2011; Hammond, D et al. 2006; Roeseler & Burns 2010; Tobacco Control Section 1998). The relationship between denormalisation and policy change is likely reciprocal whereby improved norms provide a supportive environment for policy change which can be used to further shape norms (Hammond, D et al. 2006).

Policy intervention strategies, such as the provision of anti-smoking information, warning labels, increased pricing, reduced availability, and limitations on where one can smoke, successfully changed attitudes to smoking, reducing both the injunctive and descriptive norms (Ashe, Graff & Spector 2011; Hammond, D et al. 2006). Similar strategies could be used to influence the acceptability of unhealthful foods. Examples include: educating the public on methods used to market unhealthful products; provision of information regarding the dangers of excessive unhealthful food consumption; health warnings on unhealthful food packaging (e.g., foods of low nutritional value, or high in sugar or fat); and restrictions on the availability of unhealthful food (outlet placements, opening hours, age of purchaser). Strategies intending to influence physical activity behaviour may need to focus on particular sedentary behaviours that could be replaced with physically active options. For example, social denormalisation of car usage, especially for short distances, and replacement with cycling, walking and hybrid options (e.g., walking/cycling to public transport options). Interventions intending to reduce unhealthful behaviours and promote healthful options necessitate a multi-sectoral approach,

including urban design and land use planning, the food industry and marketing practices, transport (e.g., public transport) and road design, alongside education campaigns.

9.3.1.2 Choice architecture

Choice architecture relates to how potential options can be structured to influence choices. The design of environments provide subtle clues as to their expected usage, for example, large obvious handles on doors versus push plates (Thaler, Sunstein & Balz 2013). Similarly, food pricing, placement and promotions within a supermarket (e.g., height on shelves, sugary treats at check-outs, end of aisle displays) influence consumer choices (Glanz, Bader & Iyer 2012). Food pricing can be influenced through agriculture, transport, and importation policies, "taxes" on unhealthful foods, and subsidies on fresh fruits and vegetables. In store product placements and promotions could also be influenced through policies such as incentives for meeting particular standards of shelf-space assignment, food placement and promotions. Similar ideas could be used to change the fast-food and restaurant industry to reduce meal sizes, and meal sugar and fat content. Such intervention strategies may directly influence food-related behaviour while shaping the perceived normative environment.

Choice architecture principles also relate to physical activity. The placement, number and visibility of stairs, lifts and escalators, and constraints on their usage (e.g., "skip-stop" elevators) can influence physical activity behaviour (Eves et al. 2009; Nicoll 2007; Nicoll & Zimring 2009; Olander & Eves 2011). Currently, however, building codes in Australia pay little attention to design aspects relating to health promotion such as physical activity behaviour (McGann, Jancey & Tye 2013). Hence, there is opportunity to improve health behaviours through collaboration between the disciplines of architecture and health promotion (McGann, Jancey & Tye 2013).

Though some environmental redesign strategies may be difficult and expensive, others are relatively simple and inexpensive (e.g., highlighting the position of stairs within a building and changing lift access). Regardless, the cost of not making changes is high, especially when considering future health care and economic costs that could be avoided, and the ethics of not taking action (WHO 2013). Moreover, many strategies, particularly those aiming to improve physical activity behaviours, potentially have a positive environmental impact.

By redesigning environments so that the healthful choice is the obvious and easy choice (i.e., the default choice) behaviours can be shifted, along with the perceived norm (the default option is often perceived as the 'norm') (Ashe, Graff & Spector 2011; Thaler, Sunstein & Balz 2013). Multiple policies aimed at establishing new social norms and improving people's health behaviour will likely be needed in order to impact on disease rates, with policies designed to reinforce each other (Ashe, Graff & Spector 2011; Roeseler & Burns 2010). Changing social norms will be a relatively slow process, and may take a generation or more to evolve (Ashe, Graff & Spector 2011).

9.3.2 Interventions using social norms information

In addition to policies intending to change social norms and thus health behaviour and outcomes such as those described above, social norms information can be used within health promotion strategies. Interventions have demonstrated the influence of local descriptive norms information on college campus drinking behaviours (Foxcroft et al. 2015), food intake amount and selection (Robinson, Fleming & Higgs 2014; Robinson et al. 2014), pro-environmental behaviour (Nolan et al. 2008), and online shopping product selection (Demarque et al. 2015). As discussed in Chapter 7, positively framed descriptive health-related norms information could be included in health messages, ideally with injunctive norms information to reduce the possibility of a undesirable "boomerang effect" (Schultz et al. 2007). Descriptive norms provide a normative standard from which individuals are disinclined to deviate while individual deviance may be either above or below the norm (Schultz et al. 2007). Consequently, information on local descriptive norms (e.g., current prevalence of a behaviour) can result in individuals decreasing participation in healthful behaviour (e.g., physical activity) or increasing participation in unhealthful behaviour (e.g., alcohol intake) due to the individual's desire to conform (boomerang effect). A recent study used local descriptive fruit and vegetable purchasing norms information on supermarket shopping carts including an image depicting social approval (i.e., injunctive norm) (Payne et al. 2015). The intervention resulted in increased fresh fruit and vegetable purchasing in the intervention stores as compared to the control stores.

Importantly, the research in this dissertation expressed local descriptive norms objectively, that is, aggregated from individual-level population surveillance data. It is likely that individually perceived local descriptive norms differ from objective local descriptive norms.

This has been the case for a range of other objectively measured environmental variables (e.g., see Boehmer et al. 2006; Gebel, Bauman & Owen 2009). Regarding descriptive norms, research has consistently shown that college and university students typically over-estimate the alcohol consumption of their campus colleagues (Foxcroft et al. 2015), that is, the perceived local descriptive drinking norm is typically greater than the objectively measured local descriptive norm. A recent Cochrane review on informational descriptive norms interventions aimed at reducing on-campus drinking behaviour reported beneficial effects (more accurate perception of drinking norms, reduction in binge drinking, drinking quantity and frequency) though the effect sizes were small (Foxcroft et al. 2015). Small effect sizes across populations can result in large population wide benefits by shifting the entire risk distribution curve in a beneficial direction (Rose 1985). The review findings suggest that the provision of descriptive norms information may support other intervention strategies in improving population health by correcting misperceptions of what other people do (descriptive norms). This likely represents just one of the ways in which descriptive norms information may be useful in population health initiatives. Improved understanding of what underlies the misperception between objective and perceived descriptive norms (e.g., differences according to psychosocial factors) would further support such interventions.

9.3.3 Geographically defined at-risk populations

Local descriptive norms can also be used to identify geographically defined populations at greater risk of adverse health outcomes. The growing interest in the use of GIS and spatial techniques, including geocoding of population health surveillance data, is consistent with and can support such efforts. Geographically defined populations identified as at-risk should be priorities for community health interventions. Firstly, from an individualistic perspective, these areas include a greater number of individuals identified as being ‘at risk’ (e.g., overweight/obese, physically inactive or poor diet) making the area a likely target for interventions. Secondly, from a collectivist perspective, areas with a higher unhealthful descriptive norm (compositional factor) may predispose individuals within the area to ‘adjust’ to the area-level norm. Individuals of normal weight within such a region may be at greater risk of becoming overweight (Blok et al. 2013), with the subsequent health consequences (such as worsening cardiometabolic risk; Carroll, SJ et al. 2016). This would only serve to reinforce the local descriptive unhealthful norm (i.e., increase local prevalence). Further, areas

with a greater unhealthful descriptive norm may be resistant to healthful change. For example, where a larger body size is accepted as the norm, interventions promoting weight loss and healthful dietary and physical activity behaviours are unlikely to be motivating unless the relevant social norms are also targeted. Interventions within such areas should include a focus on improving the local norm (e.g., denormalisation of the unhealthful norm) so that the 'norm' does not undermine other intervention efforts.

An additional concern is that area-level norms may worsen. Assuming a right skewed distribution, as individuals conform to the 'average BMI', the average BMI itself can be pushed upwards (Hammond, RA & Ornstein 2014). Area-level norms may also spread between areas as the influence of an area does not end at a pre-defined spatial unit boundary. Spatial influences likely reduce with distance suggesting nearby areas in particular may be at increased risk of increasing unhealthful norms (Tobler 1970). Consequently, there is a need to reduce the potential impact these at-risk areas may have in terms of spreading norms. Such areas should be priority targets for health interventions.

Both walkability and availability of healthful food resources each interacted with local descriptive norms for overweight/obesity in relation to change in HbA_{1c}. Therefore areas identified as at increased risk due to a greater descriptive norm for overweight/obesity should consider walkability and healthful food as likely targets for interventions along with efforts to change the overweight/obesity norm (and vice versa). Improving walkability or availability of healthful food within areas with a greater overweight/obesity norm could provide greater population health benefit than improving walkability or healthful food availability in areas with a low overweight/obesity norm.

9.3.4 Diffusion of Innovations

Lastly, descriptive norms information may be used to develop population targeted interventions according to the current behaviour or trait prevalence. Rogers' Diffusion of Innovations (Rogers 2003) suggests that where the local prevalence of the norm of interest is low, intervention strategies may need to target early adopters. Conversely, where the local prevalence is high, targeting laggards may be more effective. Intervention strategies will need to differ depending on the target group. Innovators and early adopters may respond to education campaigns appealing to values, attitudes and beliefs which predispose behaviour

(e.g., the benefits of improving their health behaviours), the early majority may respond to the removal of barriers (such as improving the built environment), while late majorities and laggards may be more influenced by provision of descriptive norms information (Rogers 2003).

This section has provided a spectrum of ideas on how descriptive norms can be shaped through environmental redesign, how local descriptive norms information can be used within interventions, and highlighted the need to target areas with worse local norms as they are at increased risk, likely to be resistant to healthful changes and may increase risk for individuals residing in nearby areas through the spread of unhealthful norms. To effectively reduce the risk of cardiometabolic diseases, a multi-level and multi-sectoral approach using integrated and supporting intervention strategies will be needed.

9.4 Strengths and limitations

9.4.1 Conceptual framework, and operational model

This research tested hypotheses defined within an operational model (Figure 4.4, page 65) based on an empirically derived conceptual framework (Figure 3.3, page 29). The conceptual framework was designed to demonstrate the biosocial pathways linking environments to health outcomes (Daniel et al. 2011). That is, the framework clearly expresses the linkage between environmental risk conditions and cardiometabolic risk through a variety of causal pathways. The research within this dissertation draws on this foundation to empirically test a component of the framework as outlined in the operational model.

9.4.2 Causal inference

The longitudinal design, including the use of three waves of data from a 10-year population-based biomedical cohort, is a strength of this research. Longitudinal research design supports causal inference through the temporal order of measures (Daniel, Moore & Kestens 2008). The use of a longitudinal design is an improvement over the cross-sectional studies typically conducted in research assessing associations between local environments and health outcomes. It is important to note, that environmental exposures for this research were expressed at only one time point thus precluding examination of the possibility that the levels of environmental exposures might change over time. Inclusion of environmental exposure information at multiple time-points may improve analyses so they are more reflective of the actual exposures experienced by individual residents including the approximate lengths of time for these levels of exposures.

Temporality of measures (i.e., longitudinal design) does not, on its own, provide sufficient evidence for causality (Hill 1965). Testing of the plausible biosocial pathways would further support causal inference (Daniel, Moore & Kestens 2008; Hill 1965). Though such testing was not performed, potential pathways have been clearly identified within the conceptual framework underpinning this research. In this dissertation, it is assumed that the associations between the physical activity and food environments, and change in cardiometabolic risk are mediated by individual-level physical activity and dietary behaviour, respectively.

With regard to other criteria for causation (per Hill 1965, italicised below), the findings are *coherent* in that they do not conflict with generally accepted theories and empirical findings.

Chapter 3 provided a strong theoretical argument for the need to consider environmental risk conditions, introducing local descriptive norms as potentially important environmental influences on health. This need was further supported by Chapter 4 and previous empirical observations, especially relating to the contextual built environment, and the results reported here align with theory. Though the *strength* of associations between the environmental exposure measures and change in HbA_{1c} was small in magnitude, this aligns with much of the research on associations between environmental risk conditions and health outcomes. Small effect sizes may be attributed to the distal nature of the influence, acting through multiple other influences. Importantly, though the associations may be small, the effect at the population level can be substantial (Rose 1985).

Another of Hill's criteria to be discussed here is *consistency* (Hill 1965). Walkability has consistently been reported as associated with physical activity behaviour (Hajna et al. 2015; Lovasi, Grady & Rundle 2012; Saelens & Handy 2008; Sallis et al. 2012) and health outcomes (Leal & Chaix 2011; Lovasi, Grady & Rundle 2012), further supporting causality. The investigation of local descriptive norms in relation to health is novel. Associations between the local descriptive norms and HbA_{1c} were consistent within the included studies, that is, each of the three norms tested were statistically significantly associated with change in HbA_{1c} over time. However, both Study 2 and 3 are based in the same cohort and study region.

9.4.3 Residential self-selection

Though this research was longitudinal in design, it was still observational with consequent limitations as to the conclusions that can be inferred (Oakes, Mậsse & Messer 2009). Of particular note, one must consider the potential influence of residential self-selection of individuals (or households) into preferred neighbourhoods as constrained by their opportunities (Lovasi, Grady & Rundle 2012). Individuals who value particular environmental features, for example neighbourhood walkability, may both choose to live in more walkable areas and walk more. Consequently, greater walking (and cardiometabolic health) among residents of walkable areas may not be due to the effect of walkability on behaviour directly, but due to physically active (and healthier) individuals self-selecting to walkable areas. Apparent associations between the built environment and travel behaviours such as walking, may be confounded by residential self-selection (Cao, Mokhtarian & Handy 2009). The influence of environmental exposures may, therefore, be mis-estimated.

While experimental studies could be designed to address residential self-selection within efforts to understand environmental influences on health (e.g., the Moving to Opportunity Program) (Katz, Kling & Liebman 2001; Kaufman, Kaufman & Poole 2003), such studies are rarely feasible due to ethical considerations, impracticality and cost (Cao, Mokhtarian & Handy 2009; Kaufman, Kaufman & Poole 2003). Moreover, the actualisation of residential preference for particular features will be limited by factors including the availability of housing in neighbourhoods within a price range, and other preferred features (e.g., close to work, friends or family). For residential self-selection to seriously bias results, the preference for any given environmental feature (e.g., walkability) would need to be relatively unconstrained by other considerations (e.g., socioeconomic) (Lovasi, Grady & Rundle 2012). The relative importance of environmental features, such as walkability, in relation to residential self-selection is currently unclear (Lovasi, Grady & Rundle 2012).

Studies have assessed the impact of residential self-selection on associations between the built environment and transport behaviour. One review of 38 empirical studies reported that, though there is evidence of residential self-selection, virtually all reviewed studies found a statistically significant influence of the built environment on transport behaviour (including walking) after accounting for residential self-selection (Cao, Mokhtarian & Handy 2009). In general, not accounting for residential self-selection is believed to inflate estimates of associations between the built environment and travel behaviours. However, one study found that residential self-selection can bias estimates in *either* direction, yielding over or under-estimated effects of the built environment on travel behaviours, but this bias is not very strong, typically attenuating estimated effects rather than rendering them no longer statistically significant (Chatman 2009). Regarding the Studies 2 and 3 of this dissertation, while analysis models included individual-level factors that may influence residential self-selection (e.g., individual-level SES), the included analyses could not account for, and have not directly accounted for residential self-selection. Therefore, self-selection of individuals into neighbourhoods with preferred features may have biased the observed findings.

9.4.4 Measures

The measures used in this research were explicitly defined, and their operationalisation and expression based on previously used measures or concepts and recommendations presented within the literature (i.e., local descriptive norms and cCICR). In Studies 2 and 3,

cardiometabolic risk was expressed in a continuous format using a clinical measure, HbA_{1c} concentration. The use of a clinical outcome is an improvement over the commonly used self-reported health outcomes as it avoids self-report bias. Expressing cardiometabolic risk in a continuous format provides information regarding risk severity and therefore, more precisely reflects the magnitude of change over time. The use of continuous measures has been recommended (Kahn et al. 2005) and such measures have greater statistical power than categorised measures (Ragland 1992).

Though the outcome was expressed using a clinical measure, the potential for self-report bias is not completely avoided as individual demographic and smoking data were self-reported. In addition, local descriptive norms (prevalence of overweight/obesity, physical inactivity, and insufficient fruit intake) were aggregated from self-report survey data (SAMSS) with the consequent possibility of self-report bias. Collecting information through self-report is a practical, low cost method suitable for sampling large numbers of participants (Gorber et al. 2007). However, self-report information is not always accurate due to questions being misunderstood, inaccurate recall, and response bias. For example, self-reported fruit consumption and physical activity are each susceptible to social desirability biases (Brenner & DeLamater 2014; Miller et al. 2008). A systematic review comparing direct versus self-reported height, weight and BMI concluded overall tendencies for height to be overestimated, and weight and BMI to be underestimated relative to the measured values (Gorber et al. 2007). Short food frequency questionnaires are susceptible to systematic bias (Kirkpatrick et al. 2014), and it is generally believed that physical activity is typically over-reported when using self-report methods compared with direct measures (Brenner & DeLamater 2014). However, one review reported that self-reported physical activity may over- or under-estimate physical activity compared to directly measured physical activity (e.g., doubly-labelled water, accelerometers), depending on which self-report method was used (Prince et al. 2008). If, as is commonly accepted, BMI is under-reported and physical activity and fruit intake over-reported, then the absolute values of the prevalence rates for NWAHS buffers may be under-estimated. However, *relative* comparisons of buffer prevalence rates undertaken here should not be greatly affected. As environmental exposure measures were standardised prior to inclusion in analysis models and multilevel regression methods used for

analyses, the under-estimation of absolute prevalence rates should not greatly impact estimates of associations between local descriptive norms and change in HbA_{1c}.

The methods and data sources used in operationalising environmental exposures are further strengths of this work. Local descriptive norms data were derived from a separate survey, thus avoiding same-sample bias (Diez Roux 2007). The contextual physical activity and food environments were represented using objective measures. Walkability was expressed using a measure similar to those used in Australia and internationally, per the International Physical Activity and Environment Network (IPEN). POS data were extracted from a dataset validated against historical imagery. Food environment data (food outlet locations and type) were extracted from a retail database constructed from data collected by field surveyors (DPTI 2007a). It is noted, however, that objective measures, such as those used in this research, and perceived measures often differ (Boehmer et al. 2006; Gebel, Bauman & Owen 2009). Though this research did not include perceived measures, this is not to discount their importance. A previous cross-sectional study using the NWAHS data found metabolic syndrome was negatively associated with greater perceived local land use mix, aesthetics and walking infrastructure, and negatively associated with greater perceived crime and barriers to walking (Baldock et al. 2012).

9.4.5 Spatial units

As recommended (Chaix et al. 2009), environmental exposures were defined using ego-centred road-network buffers. Area-level education was expressed in a manner intended to closely approximate these road-network buffers. In consideration of the MAUP (see Section 4.5) (Openshaw 1984; Openshaw & Taylor 1979), the use of several differently sized road network buffers to define environmental exposures has been recommended (Brownson et al. 2009; Chaix et al. 2009) and would have been ideal, however, it was not possible to include differently sized buffers for reasons previously outlined (Section 5.7). The use of differently sized units may result in differences in findings, particularly in the strength of relationships found (Coffee et al. 2013; Flowerdew, Manley & Sabel 2008; Openshaw 1984).

9.4.6 Residential environments as influences on health

A fundamental premise of this research is that individuals are influenced by their residential environments. This does not account for time spent away from local residential areas, nor

behavioural patterns pertaining to where food is purchased or physical activity performed, limitations of this dissertation. This assumption may be specious in that individuals may purchase their food from places outside of their immediate local residential area. Similarly, individuals may participate in physical activity in locations (e.g., POS) outside of their local residential area. Assessment of food purchasing habits (and physical activity behaviours), in terms of distances travelled, and reasons for selection of preferred stores (or preferred physical activity facilities such as POS) used is needed. Moreover, services exist that home deliver food, including both healthful and unhealthful food options, as either prepared meals or ingredients. These include a variety of restaurant types (e.g., pizza, Chinese, Indian etc.), meal delivery services (delivering fresh or frozen meals), fresh food deliveries from major supermarket retailers, and more recently, food delivery services that provide recipes along with the necessary fresh ingredients on a weekly basis (e.g., HelloFresh©).

Lastly, in relation to residential environments, the influence of residential environments can differ according to individual sociodemographic factors. The analyses reported here accounted for individual age as a covariate, but older individuals and those caring for young children at home may spend more time closer to home thus resulting in greater exposure and consequently stronger outcome from local environment exposures. Similarly, car ownership may modify associations between residential environments and health outcomes as car ownership provides easier, more convenient access to facilities further away from an individual's place of residence.

9.5 Implications for advancing the research field

9.5.1 Expression of cardiometabolic risk

Though the cCICR demonstrated moderate accuracy in predicting 10-year incident cardiometabolic disease, assessment of the cCICR's utility in assessing change over time indicated that the component risk factors change differentially over time. This may be due to differential responses to environmental exposures. Thus future research on the utility of cCICRs in longitudinal research is recommended prior to usage in studies assessing change in cardiometabolic risk in response to environmental exposures. However, research focusing on the differential responses of cardiometabolic risk factors in relation to environmental (and behavioural) exposures may be more informative, and should be prioritised. Such studies could assess the influence of environmental exposures on a variety of different cardiometabolic risk factors concurrently, within the same region and population. Explication of specific relations would support causal inference by clearly defining biological plausibility of pathways relating environments to health outcomes. Moreover, understanding specific relations would inform intervention strategies which could target priority risk factors.

9.5.2 Causal inference

The majority of studies regarding the influence of residential area features to health outcomes are cross-sectional in design (Leal & Chaix 2011; Mackenbach et al. 2014; Malambo et al. 2016; Schüle & Bolte 2015). Inference on the direction of causation is limited in cross-sectional studies and findings may be due to reverse causation (Diez Roux & Mair 2010; Leal & Chaix 2011; Yen, Michael & Perdue 2009). Temporality, supporting causal inference, can only be assessed using a longitudinal design (Daniel, Moore & Kestens 2008; Hill 1965). More longitudinal studies are therefore needed to provide stronger evidence to support causal relationships between features of environments and cardiometabolic outcomes.

Few studies have assessed relationships between local descriptive norms and health outcomes. Though the findings of Studies 2 and 3 suggest the importance of local descriptive norm in relation to health outcomes, more research assessing associations between local descriptive norms and health outcomes in other regions and populations is needed to establish consistency of associations between studies, supporting causal inference (Hill 1965).

In this dissertation, it is assumed that the associations between environmental features and change in cardiometabolic risk are mediated by individual-level physical activity and dietary behaviour. However, few studies have assessed mediators between built environment exposures and cardiometabolic risk. No studies have assessed mediators between local descriptive norms and cardiometabolic risk. Future studies should formally assess potential mediators (e.g., individual walking, physical activity and dietary behaviours) between compositional norms, built environment features and cardiometabolic outcomes. Such research could provide empirical evidence of pathways connecting environments and health outcomes, further supporting causal inference (Daniel, Moore & Kestens 2008; Hill 1965).

As environmental influences on individual-level health outcomes are likely due to multiple, highly interdependent pathways, complex systems approaches such as agent-based modelling may be useful to identify the complex causal processes at work (Auchincloss & Diez Roux 2008; El-Sayed et al. 2012; Hammond, RA 2009; Marshall & Galea 2015). Agent-based models are stochastic computer simulations of systems (artificial societies) consisting of a collection of "agents" placed in a spatial context. These "agents" interact and change over time giving rise to macrosystems. "Agents" are anything that alters its behaviour in response to input from other agents and the environment (Auchincloss & Diez Roux 2008; El-Sayed et al. 2012; Hammond, RA 2009). The macro-level patterns generated by the computer simulation model (e.g., changes in distribution of BMI, eating patterns, smoking behaviour, health outcomes) can be directly compared with data to calibrate the model (Hammond, RA 2009). Such models can be used to extend theory and test hypotheses about fundamental processes (Auchincloss & Diez Roux 2008). Of particular note in relation to causal inference, counterfactuals can be simulated to assess model response to change in an exposure of interest (El-Sayed et al. 2012; Marshall & Galea 2015).

In an effort to explicate the mechanism underpinning the upward trend in obesity prevalence and clustering of obesity within networks, one agent-based model focused on developing a theoretical mechanism of social influence – individual conformity to body type norms – on the development and persistence of obesity (Hammond, RA & Ornstein 2014). Testing against longitudinal data of students within 181 schools in Québec, Canada provided empirical support for this model.

9.5.3 Composition beyond area-level SES

Composition has typically been regarded as the summed influence of individual-level characteristics. Though the influence of area-level SES on health outcomes has been assessed in numerous studies, the remaining variance ascribed to area-level SES after inclusion of individual-level characteristics has typically been conceived of as reflecting a contextual influence. This notion, however, surely needs to be rethought, because it reduces compositional influences to the level of a nuisance variable to be controlled for, and prevents the explicit modelling of compositional measures as predictor variables. Composition is clearly more than a nuisance variable, representing the influence of collective lifestyles and local descriptive norms on individual-level health and behaviour. This premise is supported by the research results from Studies 2 and 3 and highlights the importance of expanding the research focus of studies to assess compositional influences, conceptualised as more than the sum of individual-level influences, and to broaden the focus beyond area-level SES.

9.5.4 Social norms

'Norms' have typically been considered within the social sphere (subjective norms), that is, as a social influence defined between individuals connected by strong affective ties such as family and friends (e.g., Theory of Planned Behaviour and Social Contagion Theory; Ajzen 1991; Christakis & Fowler 2013, respectively). This approach has ignored the influence imparted simply through sharing the same 'space', limiting the development of understanding these influences. *Local* norms, both injunctive and descriptive, should be considered in future research regarding the influence of norms on behaviour and health outcomes.

Research interested in relations between environments and health outcomes should broaden focus to consider local descriptive norms. Of particular interest would be to also include subjective descriptive norms (i.e., what family and friends do) in models assessing associations between local descriptive norms and health outcomes. This research could be further expanded to include injunctive norms and interactive effects between the resultant four norm expressions (injunctive/descriptive and local/subjective). In many situations these norms do not concur, and may even be contradictory (Lapinski & Rimal 2005). Psychology research has reported independent and interactive effects of local and subjective, descriptive and injunctive norms (Cialdini, Reno & Kallgren 1990; Fornara et al. 2011; Kallgren, Reno & Cialdini 2000; Kormos, Gifford & Brown 2015; Reno, Cialdini & Kallgren 1993; Rivis & Sheeran 2003;

Smith et al. 2012). Understanding the contribution of local descriptive norms within the context of environments and health while accounting for other supporting and contradictory normative influences could lead to more targeted intervention strategies. Research could also focus on competing definitions of 'local' such as residential and workplace norms. Such explorations may assist in informing intervention design by identifying the targets with the best cost-benefit ratio.

A further extension of this research effort would be to include assessment of potential moderators of the influence of norms. For example, the Theory of Normative Social Behaviour (Rimal & Real 2005) identifies three potential moderators of the influence of perceived descriptive norms on behaviour, namely injunctive norms, outcome expectations, and group identity. Other factors, such as individual-level sociodemographic and psychosocial factors (e.g., age, sex, self-concept and personality type) (Cialdini & Trost 1998; Leroux et al. 2012), cultural factors (independent/individualistic versus interdependent/collectivistic) (Bagozzi et al. 2000; Markus & Kitayama 1991), and attributes of the behaviour of interest (e.g., public versus private) (Cialdini & Trost 1998; Lapinski & Rimal 2005), are also likely to be important moderators of the effect of norms on health behaviour and outcomes. This dissertation makes an important contribution to research on environmental exposures and health outcomes by assessing the interactive effects of local descriptive norms and built environment features in relation to change in cardiometabolic risk.

While exploration of moderators of the influence of norms is important, such research will not improve understanding of how norms develop, are changed, or are transmitted within and between groups and locations. Developing understanding in this regard may inform intervention strategies by identifying levers upon which to act. Of particular importance may be identifying ways to limit the spread of unhealthful norms within social groups and local areas, but also between groups and areas. As norms must be communicated to have any effect on behaviour (Cialdini & Trost 1998), consideration of communication theories may be key to understanding their mechanism of influence, and methods of norms transmission.

Provision of descriptive norms information has previously been shown to influence individual intentions and behaviours (Demarque et al. 2015; Foxcroft et al. 2015; Nolan et al. 2008; Robinson, Fleming & Higgs 2014; Robinson et al. 2014) presumably by correcting

misperceptions of what other people do (descriptive norms) (Foxcroft et al. 2015). Improved understanding of what underlies the misperception between objective and perceived descriptive norms (e.g., differences according to psychosocial factors) may inform intervention strategies and further research in this area is recommended.

9.5.5 Contextual built environment measures

The measures used in Studies 2 and 3, representing the contextual physical activity and food environments, were objectively defined and selected to enable comparability with other studies. The aim of this dissertation was not to further develop research relating to the built environment *per se*, but to highlight the need for consideration of compositional local descriptive norms while accounting for the contextual built environment. To best achieve this, built environment measures that have previously been used and shown to be related to cardiometabolic risk were included in analytic models. Though it was not a focus of this dissertation, there is, however, a need to develop further the methods used in operationalising the built environment. The unexpected findings from Study 2 for POS, and the null findings for food resource availability from Study 3 highlight this point.

Though the walkability score was associated with change in cardiometabolic risk in Study 2, the actual walkability of an area likely depends on more than 'proximity' and 'connectivity'. Other built environment features may also be important such as traffic (noise, volume, speed and safety), signs of physical disorder (e.g., litter, graffiti and crime), sense of safety, presence and quality of sidewalks, lighting, trees, greenery, and natural scenery (Lovasi, Grady & Rundle 2012; Sugiyama et al. 2012). Further, the importance of each of these features may differ depending on the type or purpose (recreational versus transport) of walking or other physical activity (e.g., cycling). Similarly, availability of POS, expressed as a proportion of buffer area (POS area/area of buffer intersected parcels) ignores a multitude of other, potentially important factors such as park type, size, quality, and available facilities (Giles-Corti et al. 2005; Hillsdon et al. 2006; Jones, Hillsdon & Coombes 2009; Paquet et al. 2013). Future research, focused on the contextual built environment, should attempt to refine these definitions and assess the relative importance of the different walkability and POS related features to specific behaviours and consequent health outcomes. Studies could assess multiple aspects of 'walkability' (e.g., street connectivity and provision of footpaths) in

relation to walking behaviour, which could itself be refined to recreational or transport related, and health outcomes to determine priority targets for interventions.

Regarding the contextual food environment, in Study 3 neither fast-food nor healthful food availability were associated with change in HbA_{1c}. As food resource availability likely functions as an enabler (or barrier) to obtaining desired foods, the null findings may simply reflect sufficient access to each type of food resource within the study area. However, as with walkability and POS, improvements in the expression of food availability are recommended as a focus for future research. The ecologically based conceptual model of Glanz and colleagues (2005), presented as Figure 4.2, identifies four types of food 'environments'. Studies regarding local food environment risk conditions, similar to Study 3, typically focus on only one of these 'environments', the *community nutrition environment*. While this can be informative, such studies disregard the *consumer environment*, *organisational nutrition environment*, and *information environment*, each of which influences dietary behaviour and health outcomes. Further, these influences are likely to act in concert. An individual may be more inclined to purchase (and consume) fruit when each (or many) of these environments support such an action. That is, when fruit is locally available (community nutrition environment), of good quality and cheap in price (consumer environment), and the information environment promotes the benefits of eating fruit, individuals may be more likely to purchase and consume fruit. Future research regarding the influence of the local food environment could expand focus to include features from multiple of the 'environments' identified by Glanz and colleagues (2005). Examples could include the integration of store audits with GIS measures to represent both the community nutrition and consumer environments.

Though the research within this dissertation did not include perceived measures, this is not to discount their importance. Individual perceptions of environments are distinct from objective environmental measures though both are potentially important influences on physical activity (Baldock 2015; Sallis et al. 2006). Perceived measures reflect an individuals' interaction with the environment and may be influenced by individual (and other) factors (Gebel, Bauman & Owen 2009). An Australian study found adults with lower educational attainment, lower incomes, who were overweight or less physically active for transportation purposes were more likely to misperceive their high walkable neighbourhood as low walkable (Gebel,

Bauman & Owen 2009). Correspondence between objective and perceived measures of environmental features is typically low (Boehmer et al. 2006; Gebel, Bauman & Owen 2009). Future research could consider both objective and perceived measures, and the potential individual factors that may underlie misperceptions, to further develop understanding of the relationship between the objective and perceived environments and health outcomes.

9.5.6 Summative, interactive, and threshold effects

Much of the research regarding the influences of environmental features on health outcomes thus far, has sought to establish the importance of environments, or specific features of environments, on health outcomes. As such, they have typically focused on whether or not environmental features are associated with health outcomes. There is a need to develop this further, to assess the *shape* of such associations to determine potential thresholds for the influence of environmental exposures. Resultant information could inform policies intending to improve population health through urban design and redevelopment by providing minimum specifications for built environment features.

It is likely that built environment features function as both summative and interactive influences on individual-level behaviour and health outcomes. Environments with adequate levels of *many* relevant built environment features to a specific health behaviour (e.g., walking) will likely have a greater influence than environments with adequate levels of a few built environment features (or none). Moreover, the levels of particular environmental features may inhibit or enhance the influence of other environmental features. The findings from Studies 2 and 3 on the interactive influence of local descriptive overweight/obesity norms and walkability or healthful food availability in relation to change in HbA_{1c}, suggests this is the case. However, further research in this regard is needed to examine which summative and interactive combinations of environmental features are the most influential on health while considering the feasibility of environmental changes.

The conditions for whom, each built environment feature relates to health behaviour also need further assessment. For example, the importance different park features in relation to behaviour and health, will likely differ for the elderly (e.g., benches, shade) versus young families (e.g., playground). Understanding which features of parks are important for different groups of people could inform park (and neighbourhood) design, such that parks can be

created to attend the needs of all members of the local community. Though studies have previously reported individual-level moderation effects on the relationship between the physical activity environment and physical activity behaviour or cardiometabolic outcomes according to age and sex (e.g., Cain et al. 2014; Richardson & Mitchell 2010; Shigematsu et al. 2009), more research is needed.

9.5.7 Expressions of environments

Estimates of associations between environmental exposures and health outcomes may vary between types and sizes of spatial units used in operationalising exposures (Coffee et al. 2013; Flowerdew, Manley & Sabel 2008; Openshaw 1984). Future studies should endeavour to express environmental exposure information for a range of buffer sizes to inform on appropriate size for the exposure and outcome of interest as has previously been recommended (Brownson et al. 2009; Chaix et al. 2009).

Consideration of environmental exposures beyond residential environments is also warranted. Understanding consumer choices as to where, when and how they purchase their foods or participate in physical activity, and the reasons they make such choices, is needed so that research assessing environmental influences can focus on the appropriate environment. Further, developing an understanding of travel behaviours, exposures along travel corridors and at workplaces (or other frequently attended venues) is needed. Research could focus on 'activity living spaces' using Global Positional System (GPS) technologies, travel diaries and surveys to develop understanding of behavioural patterns (e.g., Cetateanu & Jones 2016; Kestens et al. 2010; Zenk et al. 2011). Research could include investigations of the relative contributions of different environments on health outcomes, informing which environment (e.g., work versus home) provides the greatest impact on health and is therefore a priority target for intervention.

9.6 Conclusion

Cardiometabolic risk is a growing concern worldwide. Strategies aimed at reducing cardiometabolic risk are urgently needed to impact on the increase in prevalence of cardiometabolic diseases. Interventions that target environments can have population wide impacts in reducing cardiometabolic risk and outcomes by shifting the population risk distribution in a favourable direction. It is therefore necessary to identify which mutable features of environments influence individual-level cardiometabolic outcomes. This dissertation, and the three included manuscripts, add to the body of research regarding expressions of cardiometabolic risk, and the independent and interactive influences of environmental features and in relation to change in cardiometabolic risk.

The construction and validation of the cCICR included within this dissertation provides an alternative expression of cardiometabolic risk that may be appropriate for use in empirical research needing a continuous clinical expression of cardiometabolic risk. However, further research is still needed prior to use of the cCICR within longitudinal studies. Consideration of appropriate expressions of cardiometabolic outcomes is important for methodological rigour. The follow-up study highlighted that cardiometabolic risk factors respond differentially over time, potentially due to differential responses to behavioural and environmental exposures. Therefore the use of single cardiometabolic risk factors in research seeking to understand environmental influences on cardiometabolic outcomes may be informative as to the specific pathways through which such environmental exposures act.

This dissertation introduced a novel group of environmental exposures, local descriptive norms, to research regarding the influence of place on health, providing both theoretical and empirical evidence to support their inclusion in future research. It is hoped that the published manuscripts (2 and 3) will generate enough interest that other researchers consider local descriptive norms in relation to health outcomes in other regions and populations. Consideration of the influence of local descriptive norms offers enormous potential to the development of more effective public health interventions.

This research provided further support for the importance of contextual built environment features in relation to health outcomes, particularly in regards to walkability. Moreover, these findings support the hypothesis of interactions between environmental exposures in relation

to health outcomes. This implies there is a need to consider both the independent and interactive influences of multiple environmental features simultaneously in intervention design.

This dissertation makes a valuable contribution to the field of research regarding environmental influences on health outcomes. Methodological rigour requires the consideration of the expression of cardiometabolic risk as reflected by Objective 1. Though the cCICR constructed and assessed in Study 1 may not be appropriate for use in longitudinal studies without further assessment, it may be applicable for cross-sectional studies. This dissertation also highlights the need to consider composition as more than the sum of individual influences, but as an attribute of the social collective, expressed as collective lifestyles and local norms. This work provides both theoretical and empirical support for the importance of local descriptive norms, and therefore composition as a collective attribute, in relation to cardiometabolic risk. Lastly, this work includes theoretical and empirical support for the premise of interaction effects between environmental exposures in relation to cardiometabolic risk. It is hoped that this dissertation will bring attention to local descriptive norms and environmental interaction effects so that health promotion policy and intervention design can be better informed and health for all individuals improved.

References

ABS 2001, *Statistical Geography Volume 2: Census Geographic Areas Australia*, cat.no. 2905.0, Australian Bureau of Statistics, Canberra.

ABS 2003, *Usual Residents Profile 2001*, cat.no. 2004.0, Australian Bureau of Statistics, Canberra.

ABS 2006a, *Australian Standard Geographic Classification Vol. 2: Census Geographic Areas, Australia 2006*, cat.no. 2905.0, Australian Bureau of Statistics, Canberra.

ABS 2006b, *Socio-Economic Indexes for Areas (SEIFA) - Technical Paper*, cat.no. 2039.0.55.001, Canberra.

ABS 2006c, *Basic Community Profile (BCP) DataPack*, cat.no. 20069.0.30.001 (Second Release), Australian Bureau of Statistics, Canberra.

ABS 2007, *Discover Your Census: Products and Services of the 2006 Census*, Australian Bureau of Statistics, Canberra.

ABS 2008, *Information Paper: An Introduction to Socio-Economic Indexes for Areas (SEIFA) 2006*, cat.no. 2039.0, Australian Bureau of Statistics, Canberra.

ADA 2015, '2. Classification and diagnosis of diabetes', *Diabetes Care,* vol. 38, no. S1, pp. S8-S16.

Adams, R, Howard, N, Tucker, G, Appleton, S, Taylor, A, Chittleborough, C, Gill, T, Ruffin, R & Wilson, D 2009, 'Effects of area deprivation on health risks and outcomes: a multilevel, cross-sectional, Australian population study', *International Journal of Public Health,* vol. 54, pp. 183-192.

Agarwal, S, Jacobs Jr, DR, Vaidya, D, Sibley, CT, Jorgensen, NW, Rotter, JI, Chen, YDI, Liu, Y, et al. 2012, 'Metabolic syndrome derived from principal component analysis and incident cardiovascular events: the Multi Ethnic Study of Atherosclerosis (MESA) and Health, Aging, and Body Composition (Health ABC)', *Cardiology Research and Practice,* vol. 1, no. 1, DOI: 10.1155/2012/919425.

AIHW 2010, *Australia's Health 2010: Australia's Health series no. 12*, cat.no. AUS 122, Australian Institute of Health and Welfare, Canberra.

AIHW 2012, *Australia's Health 2012: The thirteenth biennial health report of the Australian Institute of Health and Welfare*, cat.no. AUS 156, Australian Institute of Health and Welfare, Canberra.

AIHW 2013, *National Health Priority Areas*, Australian Institute of Health and Welfare, Canberra, viewed 13/12/2013, <http://www.aihw.gov.au/national-health-priority-areas/>.

AIHW 2014a, *Cardiovascular disease, diabetes and chronic kidney disease—Australian facts: morbidity–hospital care*, cat.no. CDK 3, Australian Institute of Health and Welfare, Canberra.

AIHW 2014b, *Cardiovascular disease, diabetes and chronic kidney disease—Australian facts: mortality*, cat.no. CDK 1, Australian Institute of Health and Welfare, Canberra.

AIHW 2014c, *Cardiovascular disease, diabetes and chronic kidney disease—Australian facts: prevalence and incidence*, cat.no. CDK 2, Australian Institute of Health and Welfare, Canberra.

AIHW 2014d, *Australia's health 2014*, cat.no. AUS 178., Australian Institute of Health and Welfare, Canberra.

Ajzen, I 1991, 'The theory of planned behavior', *Organizational Behavior and Decision Processes,* vol. 50, pp. 179-211.

Alter, DA & Eny, K 2005, 'The relationship between the supply of fast-food chains and cardiovascular outcomes', *Canadian Journal of Public Health / Revue Canadienne de Sante'e Publique,* vol. 96, no. 3, pp. 173-177.

Ashe, M, Graff, S & Spector, C 2011, 'Changing places: policies to make a healthy choice the easy choice', *Public Health,* vol. 125, no. 12, pp. 889-895.

Astell-Burt, T, Feng, X & Kolt, GS 2014, 'Is neighborhood green space associated with a lower risk of type 2 diabetes? Evidence from 267,072 Australians', *Diabetes Care,* vol. 37, no. 1, pp. 197-201.

ATPIII 2002, 'Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report', *Circulation,* vol. 106, no. 25, pp. 3143-3421.

Auchincloss, AH & Diez Roux, AV 2008, 'A new tool for epidemiology: the usefulness of dynamic-agent models in understanding place effects on health', *American Journal of Epidemiology,* vol. 168, no. 1, pp. 1-8.

Auchincloss, AH, Diez Roux, AV, Mujahid, MS, Shen, M, Bertoni, A & Carnethon, M 2009, 'Neighborhood resources for physical activity and healthy foods and incidence of type 2 diabetes mellitus: the multi-ethnic study of Atherosclerosis', *Archives of Internal Medicine,* vol. 169, no. 18, pp. 1698-1704.

Auger, N, Daniel, M, Platt, R, Wu, Y, Luo, Z-C & Choiniere, R 2008, 'Association between perceived security of the neighbourhood and small-for-gestational-age birth', *Paediatric and Perinatal Epidemiology,* vol. 22, pp. 467-477.

Australian Government Department of Health 2014, *Australia's Physical Activity and Sedentary Behaviour Guidelines*, updated 10 July 2014, Department of Health, Australian Government, Canberra, viewed 7/10/2015, <http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-phys-act-guidelines>.

Bagozzi, RP, Wong, N, Abe, S & Bergami, M 2000, 'Cultural and situational contingencies and the Theory of Reasoned Action: application to fast food restaurant consumption', *Journal of Consumer Psychology,* vol. 9, no. 2, pp. 97-106.

Baldock, KL, Paquet, C, Howard, N, Coffee, N, Hugo, G, Taylor, A, Adams, R & Daniel, M 2012, 'Associations between resident perceptions of the local residential environment and metabolic syndrome', *Journal of Environmental and Public Health,* vol. 2012, DOI:10.1155/2012/589409.

Baldock, KL 2015, 'Neighbourhood environment and cardiometabolic risk: perception and reality', Doctor of Philosphy thesis, School of Health Sciences, Division of Health Sciences, University of South Australia, Adelaide.

Bandura, A 1971, *Social Learning Theory*, General Learning Press, New York City.

Bateman, LA, Slentz, CA, Willis, LH, Shields, AT, Piner, LW, Bales, CW, Houmard, JA & Kraus, WE 2011, 'Comparison of aerobic versus resistance exercise training effects on metabolic syndrome (from the Studies of a Targeted Risk Reduction Intervention Through Defined Exercise - STRRIDE-AT/RT)', *American Journal of Cardiology,* vol. 108, no. 6, pp. 838-844.

Bauman, AE, Reis, RS, Sallis, JF, Wells, JC, Loos, RJF & Martin, BW 2012, 'Correlates of physical activity: why are some people physically active and others not?', *The Lancet,* vol. 380, no. 9838, pp. 258-271.

Bennett, CM, Guo, M & Dharmage, SC 2007, 'HbA1c as a screening tool for detection of Type 2 diabetes: a systematic review', *Diabetic Medicine,* vol. 24, no. 4, pp. 333-343.

Benowitz, NL 2003, 'Cigarette smoking and cardiovascular disease: pathophysiology and implications for treatment', *Progress in Cardiovascular Diseases,* vol. 46, no. 1, pp. 91-111.

Berkowitz, L 1972, 'Social norms, feelings, and other factors affecting helping and altruism', in L Berkowitz (ed), *Advances in Experimental Social Psychology*, vol. 6, Academic Press, San Diego, CA, pp. 63-108.

Bernard, P, Charafeddine, R, Frohlich, KL, Daniel, M, Kestens, Y & Potvin, L 2007, 'Health inequalities and place: a theoretical conception of neighbourhood', *Social Science and Medicine,* vol. 65, no. 9, pp. 1839-1852.

Berry, TR, Spence, JC, Blanchard, C, Cutumisu, N, Edwards, J & Nykiforuk, C 2010, 'Changes in BMI over 6 years: the role of demographic and neighborhood characteristics', *International Journal of Obesity,* vol. 34, no. 8, pp. 1275-1283.

Bertrais, S, Beyeme-Ondoua, J-P, Czernichow, S, Galan, P, Hercberg, S & Oppert, J-M 2005, 'Sedentary behaviors, physical activity, and metabolic syndrome in middle-aged French subjects', *Obesity Research,* vol. 13, no. 5, pp. 936-944.

Black, C, Moon, G & Baird, J 2014, 'Dietary inequalities: what is the evidence for the effect of the neighbourhood food environment?', *Health and Place,* vol. 27, pp. 229-242.

Blanton, H & Christie, C 2003, 'Deviance regulation: a theory of action and identity', *Review of General Psychology,* vol. 7, no. 2, pp. 115-149.

Blok, DJ, de Vlas, SJ, van Empelen, P, Richardus, JH & van Lenthe, FJ 2013, 'Changes in smoking, sports participation and overweight: does neighborhood prevalence matter?', *Health and Place,* vol. 23, pp. 33-38.

Boardman, JD, Jarron, MSO, Rogers, RG & Denney, JT 2005, 'Race differentials in obesity: the impact of place', *Journal of Health and Social Behavior,* vol. 46, no. 3, pp. 229-243.

Bodicoat, DH, O'Donovan, G, Dalton, AM, Gray, LJ, Yates, T, Edwardson, C, Hill, S, Webb, DR, et al. 2014, 'The association between neighbourhood greenspace and type 2 diabetes in a large cross-sectional study', *BMJ Open,* vol. 4, no. 12, DOI:10.1136/bmjopen-2014-006076.

Bodicoat, DH, Carter, P, Comber, A, Edwardson, C, Gray, LJ, Hill, S, Webb, D, Yates, T, et al. 2015, 'Is the number of fast-food outlets in the neighbourhood related to screen-detected type 2 diabetes mellitus and associated risk factors?', *Public Health Nutrition,* vol. 18, no. 9, pp. 1698-1705.

Bodor, JN, Rose, D, Farley, TA, Swalm, C & Scott, SK 2008, 'Neighbourhood fruit and vegetable availability and consumption: the role of small food stores in an urban environment', *Public Health Nutrition,* vol. 11, no. 4, pp. 413-420.

Boehmer, TK, Hoehner, CM, Wyrwich, KW, Ramirez, LB & Brownson, RC 2006, 'Correspondence between perceived and observed measures of neighborhood environmental supports for physical activity', *Journal of Physical Activity and Health,* vol. 3, no. 1, p. 22.

Bohannon, RW 1997, 'Comfortable and maximum walking speed of adults aged 20—79 years: reference values and determinants', *Age and Ageing,* vol. 26, no. 1, pp. 15-19.

Booth, GL, Creatore, MI, Moineddin, R, Gozdyra, P, Weyman, JT, Matheson, FI & Glazier, RH 2013, 'Unwalkable neighborhoods, poverty, and the risk of diabetes among recent immigrants to Canada compared with long-term residents', *Diabetes Care,* vol. 36, no. 2, pp. 302-308.

Borsari, B & Carey, KB 2001, 'Peer influences on college drinking: a review of the research', *Journal of Substance Abuse,* vol. 13, no. 4, pp. 391-424.

Boulé, NG, Haddad, E, Kenny, GP, Wells, GA & Sigal, RJ 2001, 'Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials', *Journal of the American Medical Association,* vol. 286, no. 10, pp. 1218-1227.

Bowman, SA & Vinyard, BT 2004, 'Fast food consumption of U.S. adults: impact on energy and nutrient intakes and overweight status', *Journal of the American College of Nutrition,* vol. 23, no. 2, pp. 163-168.

Brenner, PS & DeLamater, JD 2014, 'Social desirability bias in self-reports of physical activity: is an exercise identity the culprit?', *Social Indicators Research,* vol. 117, no. 2, pp. 489-504.

Bronfenbrenner, U 1977, 'Toward an experimental ecology of human development', *American Psychologist,* vol. 32, no. 7, pp. 513-531.

Brown, W, Bauman, A, Bull, F & Burton, NW 2012, *Development of Evidence-based Physical Activity Recomendations for Adults (18-64 years)*, Australian Government Department of Health, Canberra.

Brownson, RC, Hoehner, CM, Day, K, Forsyth, A & Sallis, JF 2009, 'Measuring the built environment for physical activity: state of the science', *American Journal of Preventive Medicine,* vol. 36, no. 4, Supplement, pp. S99-S123.e112.

Burstein, R, Epstein, Y, Shapiro, Y, Charuzi, I & Karnieli, E 1990, 'Effect of an acute bout of exercise on glucose disposal in human obesity', *Journal of Applied Physiology,* vol. 69, no. 1, pp. 299-304.

Cain, KL, Millstein, RA, Sallis, JF, Conway, TL, Gavand, KA, Frank, LD, Saelens, BE, Geremia, CM, et al. 2014, 'Contribution of streetscape audits to explanation of physical activity in four age groups based on the Microscale Audit of Pedestrian Streetscapes (MAPS)', *Social Science and Medicine,* vol. 116, pp. 82-92.

Camhi, SM, Stefanick, ML, Katzmarzyk, PT & Young, DR 2010, 'Metabolic syndrome and changes in body fat from a low-fat diet and/or exercise randomized controlled trial', *Obesity,* vol. 18, no. 3, pp. 548-554.

Cao, X, Mokhtarian, PL & Handy, SL 2009, 'Examining the impacts of residential self-selection on travel behaviour: a focus on empirical findings', *Transport Reviews,* vol. 29, no. 3, pp. 359-395.

Carlson Gielen, A, McDonald, EM, Gary, TL & Bone, LR 2008, 'Using the PRECEDE-PROCEED Model to apply health behaviour theories', in K Glanz, B Rimer & K Viswanath (eds), *Health Behaviour and Health Education: Theory, Research and Practice*, 4 edn, Jossey-Bass, San Fransisco.

Carlson, JA, Sallis, JF, Conway, TL, Saelens, BE, Frank, LD, Kerr, J, Cain, KL & King, AC 2012, 'Interactions between psychosocial and built environment factors in explaining older adults' physical activity', *Preventive Medicine,* vol. 54, no. 1, pp. 68-73.

Carroll, S & Dudfield, M 2004, 'What is the relationship between exercise and metabolic abnormalities?', *Sports Medicine,* vol. 34, no. 6, pp. 371-418.

Carroll, SJ, Paquet, C, Howard, N, Adams, R, Taylor, A & Daniel, M 2014, 'Validation of continuous clinical indices of cardiometabolic risk in a cohort of Australian adults', *BMC Cardiovascular Disorders,* vol. 14, no. 1, p. 27.

Carroll, SJ, Paquet, C, Howard, NJ, Coffee, NT, Taylor, AW, Niyonsenga, T & Daniel, M 2016, 'Local descriptive norms for overweight/obesity and physical inactivity, features of the built environment, and 10-year change in glycosylated haemoglobin in an Australian population-based biomedical cohort', *Social Science and Medicine,* vol. 166, pp. 233-243.

Carrus, G, Bonnes, M, Fornara, F, Passafaro, P & Tronu, G 2009, 'Planned behavior and “local” norms: an analysis of the space-based aspects of normative ecological behavior', *Cognitive Processing,* vol. 10, no. 2, pp. 198-200.

Caspi, CE, Sorensen, G, Subramanian, SV & Kawachi, I 2012, 'The local food environment and diet: a systematic review', *Health and Place,* vol. 18, no. 5, pp. 1172-1187.

CDC 2004, *The health consequences of smoking: a report of the Surgeon General*, Centers for Disease Control and Prevention (US), Atlanta, GA.

CDC 2010, *How tobacco smoke causes disease: the biology and behavioral basis for smoking-attributable disease: a report of the Surgeon General*, Centers for Disease Control and Prevention (US), Atlanta, GA.

CDC 2015, *Diabetes Report Card 2014*, Centers for Disease Control and Prevention (US), Atlanta, GA.

Cerin, E, Saelens, BE, Sallis, JF & Frank, LD 2006, 'Neighborhood Environment Walkability Scale: validity and development of a short form', *Medicine and Science in Sports and Exercise,* vol. 38, no. 9, pp. 1682-1691.

Cerin, E, Leslie, E, Owen, N & Bauman, A 2008, 'An Australian version of the Neighborhood Environment Walkability Scale: validity evidence', *Measurement in Physical Education and Exercise Science,* vol. 12, no. 1, pp. 31-51.

Cerin, E, Leslie, E & Owen, N 2009, 'Explaining socio-economic status differences in walking for transport: an ecological analysis of individual, social and environmental factors', *Social Science and Medicine,* vol. 68, no. 6, pp. 1013-1020.

Cervero, R & Kockelman, K 1997, 'Travel demand and the 3Ds: density, diversity, and design', *Transportation Research Part D: Transport and Environment,* vol. 2, no. 3, pp. 199-219.

Cetateanu, A & Jones, A 2016, 'How can GPS technology help us better understand exposure to the food environment? A systematic review', *SSM - Population Health,* vol. 2, pp. 196-205.

Chaix, B, Lindström, M, Rosvall, M & Merlo, J 2008, 'Neighbourhood social interactions and risk of acute myocardial infarction', *Journal of Epidemiology and Community Health,* vol. 62, no. 1, pp. 62-68.

Chaix, B 2009, 'Geographic life environments and coronary heart disease: a literature review, theoretical contributions, methodological updates, and a research agenda', *Annual Review of Public Health,* vol. 30, pp. 81-105.

Chaix, B, Merlo, J, Evans, D, Leal, C & Havard, S 2009, 'Neighbourhoods in eco-epidemiologic research: delimiting personal exposure areas. A response to Riva, Gauvin, Apparicio and Brodeur', *Social Science and Medicine,* vol. 69, no. 9, pp. 1306-1310.

Chaix, B, Leal, C & Evans, D 2010, 'Neighborhood level confounding in epidemiological studies: unavoidable challanges, uncertain solutions', *Epidemiology,* vol. 21, no. 1, pp. 124-127.

Charreire, H, Casey, R, Salze, P, Simon, C, Chaix, B, Banos, A, Badariotti, D, Weber, C & Oppert, J-M 2010, 'Measuring the food environment using geographical information systems: a methodological review', *Public Health Nutrition,* vol. 13, no. 11, pp. 1773-1785.

Chatman, DG 2009, 'Residential choice, the built environment, and nonwork travel: evidence using new data and methods', *Environment and Planning A,* vol. 41, no. 5, pp. 1072-1089.

Cheung, SF, Chan, DK-S & Wong, ZS-Y 1999, 'Reexamining the Theory of Planned Behavior in understanding wastepaper recycling', *Environment and Behavior,* vol. 31, no. 5, pp. 587-612.

Christakis, NA & Fowler, JH 2013, 'Social contagion theory: examining dynamic social networks and human behavior', *Statistics in Medicine,* vol. 32, no. 4, pp. 556-577.

Christian, H, Giles-Corti, B, Knuiman, M, Timperio, A & Foster, S 2011, 'The influence of the built environment, social environment and health behaviors on body mass index. Results from RESIDE', *Preventive Medicine,* vol. 53, no. 1–2, pp. 57-60.

Cialdini, RB, Reno, RR & Kallgren, CA 1990, 'A focus theory of normative conduct: recycling the concept of norms to reduce littering in public places', *Journal of Personality and Social Psychology,* vol. 58, no. 6, pp. 1015-1026.

Cialdini, RB & Trost, MR 1998, 'Social influence: social norms, conformity and compliance', in DT Gilbert, ST Fiske & G Lindzey (eds), *The Handbook of Social Psychology*, 4 edn, vol. 1 and 2, McGraw-Hill, New York, pp. 151-192.

Cialdini, RB, Demaine, LJ, Sagarin, BJ, Barrett, DW, Rhoads, K & Winter, PL 2006, 'Managing social norms for persuasive impact', *Social Influence,* vol. 1, no. 1, pp. 3-15.

Clifton, KJ, Livi Smith, AD & Rodriguez, D 2007, 'The development and testing of an audit for the pedestrian environment', *Landscape and Urban Planning,* vol. 80, no. 1–2, pp. 95-110.

Cockerham, WC, Abel, T & Lüschen, G 1993, 'Max Weber, formal rationality, and health lifestyles', *The Sociological Quarterly,* vol. 34, no. 3, pp. 413-428.

Cockerham, WC, Rütten, A & Abel, T 1997, 'Conceptualizing contemporary health lifestyles', *Sociological Quarterly,* vol. 38, no. 2, pp. 321-342.

Cockerham, WC 2005, 'Health lifestyle theory and the convergence of agency and structure', *Journal of Health and Social Behavior,* vol. 46, no. 1, pp. 51-67.

Coffee, NT 2005, 'Constructing an objective index of walkability', Masters thesis, Discipline of Arts, Geography and Environmental Studies, University of Adelaide, Adelaide.

Coffee, NT 2013, 'Place in health', Doctor of Philosophy thesis, Discipline of Geography, Environment and Population, The University of Adelaide, Adelaide.

Coffee, NT, Howard, N, Paquet, C, Hugo, G & Daniel, M 2013, 'Is walkability associated with a lower cardiometabolic risk?', *Health and Place,* vol. 21, pp. 163-169.

Conroy, RM, Pyörälä, K, Fitzgerald, AP, Sans, S, Menotti, A, De Backer, G, De Bacquer, D, Ducimetière, P, et al. 2003, 'Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project', *European Heart Journal,* vol. 24, no. 11, pp. 987-1003.

Cooper, AJ, Forouhi, NG, Ye, Z, Buijsse, B, Arriola, L, Balkau, B, Barricarte, A, Beulens, JWJ, et al. 2012, 'Fruit and vegetable intake and type 2 diabetes: EPIC-InterAct prospective study and meta-analysis', *European Journal of Clinical Nutrition,* vol. 66, no. 10, pp. 1082-1092.

Craig, M, Twigg, S, Donaghue, K, Cheung, N, Cameron, F, Conn, J, Jenkins, A, Silink, M & for the Australian Type 1 Diabetes Guidelines Expert Advisory Group 2011, *National evidence-based clinical care guidelines for type 1 diabetes in children, adolescents and adults*, Australian Government Department of Health and Aging, Canberra.

Cummins, S, Curtis, S, Diez-Roux, AV & Macintyre, S 2007, 'Understanding and representing 'place' in health research: a relational approach', *Social Science and Medicine,* vol. 65, no. 9, pp. 1825-1838.

D'Agostino, RB, Vasan, RS, Pencina, MJ, Wolf, PA, Cobain, M, Massaro, JM & Kannel, WB 2008, 'General cardiovascular risk profile for use in primary care', *Circulation,* vol. 117, no. 6, pp. 743-753.

Daker, M 2014, 'Defining open space - not a walk in the park', Honours thesis, School of Natural and Built Environment, University of South Australia, Adelaide.

Dal Grande, E, Taylor, A, Hurst, B, Kenny, B & Catcheside, B 2004, *The Health Status of People Living in the South Australian Divisions of General Practice: South Australian Monitoring and Surveillance System July 2002 - December 2003*, Population Research and Outcome Studies Unit, South Australian Department of Health, Adelaide.

Daniel, M & Green, LW 2002, 'Health promotion and education', in L Breslow (ed), *Encyclopedia of Public Health*, vol. 2, Macmillan, New York, pp. 541-548.

Daniel, M, Moore, S & Kestens, Y 2008, 'Framing the biosocial pathways underlying associations between place and cardiometabolic disease', *Health and Place,* vol. 14, no. 2, pp. 117-132.

Daniel, M, Lekkas, P & Cargo, M 2010, 'Environments and cardiometabolic diseases in Aboriginal populations', *Heart, Lung and Circulation,* vol. 19, no. 5/6, pp. 306-315.

Daniel, M, Lekkas, P, Cargo, M, Stankov, I & Brown, A 2011, 'Environmental risk conditions and pathways to cardiometabolic diseases in Indigenous populations', *Annual Review of Public Health,* vol. 32, pp. 327-347.

Dattilo, AM & Kris-Etherton, PM 1992, 'Effects of weight reduction on blood lipids and lipoproteins: a meta-analysis', *American Journal of Clinical Nutrition,* vol. 56, no. 2, pp. 320-328.

De Koninck, M & Pampalon, R 2007, 'Living environments and health at the local level: the case of three localities in the Québec City region', *Canadian Journal of Public Health / Revue Canadienne de Sante'e Publique,* vol. 98, no. S1, pp. S45-S53.

De Meester, F, Van Dyck, D, De Bourdeaudhuij, I, Deforche, B, Sallis, JF & Cardon, G 2012, 'Active living neighborhoods: is neighborhood walkability a key element for Belgian adolescents?', *BMC Public Health,* vol. 12, no. 1, DOI: 10.1186/1471-2458-12-7.

Defries, RS & Townshend, JRG 1994, 'NDVI-derived land cover classifications at a global scale', *International Journal of Remote Sensing,* vol. 15, no. 17, pp. 3567-3586.

Demarque, C, Charalambides, L, Hilton, DJ & Waroquier, L 2015, 'Nudging sustainable consumption: the use of descriptive norms to promote a minority behavior in a realistic online shopping environment', *Journal of Environmental Psychology,* vol. 43, pp. 166-174.

DeMers, MN 2009, *Fundamentals of Geographical Information Systems*, 4th edn, John Wiley and Sons, Hoboken, NJ.

Després, JP 2008, 'CVD risk assessment: do we need the metabolic syndrome or better global cardiometabolic risk calculators?', *International Journal of Obesity,* vol. 32, no. S2, pp. S1-S4.

Deutsch, M & Gerard, HB 1955, 'A study of normative and informational social influences upon individual judgment', *The Journal of Abnormal and Social Psychology,* vol. 51, no. 3, pp. 629-636.

Dhingra, R, Sullivan, L, Jacques, PF, Wang, TJ, Fox, CS, Meigs, JB, D'Agostino, RB, Gaziano, JM & Vasan, RS 2007, 'Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community', *Circulation,* vol. 116, no. 5, pp. 480-488.

Diez Roux, AV 2001, 'Investigating neighborhood and area effects on health', *American Journal of Public Health,* vol. 91, no. 11, pp. 1783-1789.

Diez Roux, AV 2002, 'A glossary for multilevel analysis', *Journal of Epidemiology and Community Health,* vol. 56, no. 8, pp. 588-594.

Diez Roux, AV 2007, 'Neighborhoods and health: where are we and where do we go from here?', *Revue d'Epidémiologie et de Santé Publique,* vol. 55, no. 1, pp. 13-21.

Diez Roux, AV & Mair, C 2010, 'Neighborhoods and health', *Annals of the New York Academy of Sciences,* vol. 1186, no. 1, pp. 125-145.

DPTI 2007a, *South Australian Retail Database*, Department of Planning, Transport and Infrastructure, Government of South Australia, Adelaide, South Australia.

DPTI 2007b, *South Australian Property Cadastre*, Department of Planning, Transport and Infrastructure, Government of South Australia, Adelaide, South Australia.

Duffey, KJ, Gordon-Larsen, P, Steffen, LM, Jacobs Jr, DR & Popkin, BM 2010, 'Drinking caloric beverages increases the risk of adverse cardiometabolic outcomes in the Coronary Artery Risk Development in Young Adults (CARDIA) study', *American Journal of Clinical Nutrition,* vol. 92, no. 4, pp. 954-959.

Duncan, C, Jones, K & Moon, G 1999, 'Smoking and deprivation: are there neighbourhood effects?', *Social Science and Medicine,* vol. 48, no. 4, pp. 497-505.

Durand, CP, Andalib, M, Dunton, GF, Wolch, J & Pentz, MA 2011, 'A systematic review of built environment factors related to physical activity and obesity risk: implications for smart growth urban planning', *Obesity Reviews,* vol. 12, no. 5, pp. e173-e182.

Durstine, JL, Grandjean, P, Davis, P, Ferguson, M, Alderson, N & DuBose, K 2001, 'Blood lipid and lipoprotein adaptations to exercise', *Sports Medicine,* vol. 31, no. 15, pp. 1033-1062.

Eeg-Olofsson, K, Cederholm, J, Nilsson, PM, Zethelius, B, Svensson, AM, Gudbjörnsdóttir, S & Eliasson, B 2010, 'New aspects of HbA1c as a risk factor for cardiovascular diseases in type 2 diabetes: an observational study from the Swedish National Diabetes Register (NDR)', *Journal of Internal Medicine,* vol. 268, no. 5, pp. 471-482.

El-Sayed, AM, Scarborough, P, Seemann, L & Galea, S 2012, 'Social network analysis and agent-based modeling in social epidemiology', *Epidemiologic Perspectives and Innovations,* vol. 9, pp. 1-1.

Ellaway, A, Macintyre, S & Bonnefoy, X 2005, 'Graffitti, greenery, and obesity in adults: secondary analysis of European cross-sectional survey', *British Medical Journal,* vol. 331, pp. 611-612.

Eves, FF, Olander, EK, Nicoll, G, Puig-Ribera, A & Griffin, C 2009, 'Increasing stair climbing in a train station: the effects of contextual variables and visibility', *Journal of Environmental Psychology,* vol. 29, no. 2, pp. 300-303.

Ewing, G 2001, 'Altruistic, egoistic, and normative effects on curbside recycling', *Environment and Behavior,* vol. 33, no. 6, pp. 733-764.

Ewing, R & Handy, S 2009, 'Measuring the unmeasurable: urban design qualities related to walkability', *Journal of Urban Design,* vol. 14, no. 1, pp. 65-84.

Eyler, AA, Brownson, RC, Bacak, SJ & Housemann, RA 2003, 'The epidemiology of walking for physical activity in the United States', *Medicine and Science in Sports and Exercise,* vol. 35, no. 9, pp. 1529-1536.

Fagard, R 2005, 'Effects of exercise, diet and their combination on blood pressure', *Journal of Human Hypertension,* vol. 19, pp. s20-24.

Fishbein, M & Ajzen, I 1975, *Belief, Attitude, Intention, and Behavior: an Introduction to Theory and Research*, Addison-Wesley, Reading, MA.

Fishbein, M 2008, 'A reasoned action approach to health promotion', *Medical Decision Making,* vol. 28, no. 6, pp. 834-844.

Flowerdew, R, Manley, DJ & Sabel, CE 2008, 'Neighbourhood effects on health: does it matter where you draw the boundaries?', *Social Science and Medicine,* vol. 66, no. 6, pp. 1241-1255.

Forman, JP, Stampfer, MJ & Curhan, GC 2009, 'Diet and lifestyle risk factors associated with incident hypertension in women', *Journal of the American Medical Association,* vol. 302, no. 4, pp. 401-411.

Fornara, F, Carrus, G, Passafaro, P & Bonnes, M 2011, 'Distinguishing the sources of normative influence on proenvironmental behaviors: the role of local norms in household waste recycling', *Group Processes and Intergroup Relations,* vol. 14, no. 5, pp. 623-635.

Fotheringham, AS & Wong, DWS 1991, 'The modifiable areal unit problem in multivariate statistical analysis', *Environment and Planning A,* vol. 23, no. 7, pp. 1025-1044.

Foxcroft, D, Moreira, M, Almeida Santimano, N & Smith, L 2015, 'Social norms information for alcohol misuse in university and college students', *Cochrane Database of Systematic Reviews*, no. 1, DOI: 10.1002/14651858.CD006748.pub3.

Frank, LD, Engelke, P & Schmid, T 2003, *Health and Community Design: The Impact of the Built Environment on Physical Activity*, Island Press, Washington, DC.

Frank, LD, Andresen, MA & Schmid, TL 2004, 'Obesity relationships with community design, physical activity, and time spent in cars', *American Journal of Preventive Medicine,* vol. 27, no. 2, pp. 87-96.

Frank, LD, Schmid, TL, Sallis, JF, Chapman, J & Saelens, BE 2005, 'Linking objectively measured physical activity with objectively measured urban form: findings from SMARTRAQ', *American Journal of Preventive Medicine,* vol. 28, no. 2, Supplement 2, pp. 117-125.

Frank, LD, Sallis, JF, Conway, TL, Chapman, JE, Saelens, BE & Bachman, W 2006, 'Many pathways from land use to health: associations between neighborhood walkability and active transportation, body mass index, and air quality', *Journal of the American Planning Association,* vol. 72, no. 1, pp. 75-87.

Frank, LD, Sallis, JF, Saelens, BE, Leary, L, Cain, K, Conway, TL & Hess, PM 2010, 'The development of a walkability index: application to the Neighborhood Quality of Life Study', *British Journal of Sports Medicine,* vol. 44, no. 13, pp. 924-933.

French, SA, Harnack, L & Jeffery, RW 2000, 'Fast food restaurant use among women in the Pound of Prevention study: dietary, behavioral and demographic correlates', *International Journal of Obesity,* vol. 24, no. 10, pp. 1353-1359.

Frohlich, KL & Potvin, L 1999, 'Collective lifestyles as the target for health promotion', *Canadian Journal of Public Health/Revue Canadienne de Sante'e Publique,* vol. 90, pp. S11-S14.

Frohlich, KL 2000, 'The collective lifestyles framework: a contextual analysis of social practices, social structure and disease', Doctor of Philosophy thesis, Department de medicine sociale et preventive, Faculte de medecine, Universite de Montreal.

Frohlich, KL, Corin, E & Potvin, L 2001, 'A theoretical proposal for the relationship between context and disease', *Sociology of Health and Illness,* vol. 23, no. 6, pp. 776-797.

Gascon, M, Triguero-Mas, M, Martínez, D, Dadvand, P, Rojas-Rueda, D, Plasència, A & Nieuwenhuijsen, MJ 2016, 'Residential green spaces and mortality: a systematic review', *Environment International,* vol. 86, pp. 60-67.

Gauvin, L, Robitaille, É, Riva, M, McLaren, L, Dassa, C & Potvin, L 2007, 'Conceptualizing and operationalizing neighbourhoods: the conundrum of identifying territorial units', *Canadian Journal of Public Health/Revue Canadienne de Sante'e Publique*, pp. S18-S26.

Gebel, K, Bauman, A & Owen, N 2009, 'Correlates of non-concordance between perceived and objective measures of walkability', *Annals of Behavioral Medicine,* vol. 37, no. 2, pp. 228-238.

Gibbs, JP 1981, *Norms, Deviance, and Social Control: Conceptual Matters*, Elsevier, New York.

Giles-Corti, B & Donovan, RJ 2002, 'Socioeconomic status differences in recreational physical activity levels and real and perceived access to a supportive physical environment', *Preventive Medicine,* vol. 35, no. 6, pp. 601-611.

Giles-Corti, B, Broomhall, MH, Knuiman, M, Collins, C, Douglas, K, Ng, K, Lange, A & Donovan, RJ 2005, 'Increasing walking: How important is distance to, attractiveness, and size of public open space?', *American Journal of Preventive Medicine,* vol. 28, no. 2, Supplement 2, pp. 169-176.

Giskes, K, Turrell, G, van Lenthe, FJ, Brug, J & Mackenbach, JP 2006a, 'A multilevel study of socio-economic inequalities in food choice behaviour and dietary intake among the Dutch population: the GLOBE study', *Public Health Nutrition,* vol. 9, no. 1, pp. 75-83.

Giskes, K, van Lenthe, FJ, Turrell, G, Brug, J & Mackenbach, JP 2006b, 'Smokers living in deprived areas are less likely to quit: a longitudinal follow-up', *Tobacco Control,* vol. 15, no. 6, pp. 485-488.

Giskes, K, van Lenthe, F, Avendano-Pabon, M & Brug, J 2011, 'A systematic review of environmental factors and obesogenic dietary intakes among adults: are we getting closer to understanding obesogenic environments?', *Obesity Reviews,* vol. 12, no. 5, pp. e95-e106.

Glanz, K, Sallis, JF, Saelens, BE & Frank, LD 2005, 'Healthy nutrition environments: concepts and measures', *American Journal of Health Promotion,* vol. 19, no. 5, pp. 330-333.

Glanz, K, Sallis, JF, Saelens, BE & Frank, LD 2007, 'Nutrition Environment Measures Survey in Stores (NEMS-S): development and evaluation', *American Journal of Preventive Medicine,* vol. 32, no. 4, pp. 282-289.

Glanz, K, Bader, MDM & Iyer, S 2012, 'Retail grocery store marketing strategies and obesity: an integrative review', *American Journal of Preventive Medicine,* vol. 42, no. 5, pp. 503-512.

Goldstein, Noah J, Cialdini, Robert B & Griskevicius, V 2008, 'A room with a viewpoint: using social norms to motivate environmental conservation in hotels', *Journal of Consumer Research,* vol. 35, no. 3, pp. 472-482.

Goodpaster, BH, Kelley, DE, Wing, RR, Meier, A & Thaete, FL 1999, 'Effects of weight loss on regional fat distribution and insulin sensitivity in obesity', *Diabetes,* vol. 48, no. 4, pp. 839-847.

Gorber, SC, Tremblay, M, Moher, D & Gorber, B 2007, 'A comparison of direct vs. self-report measures for assessing height, weight and body mass index: a systematic review', *Obesity Reviews,* vol. 8, no. 4, pp. 307-326.

Grafova, IB, Freedman, V, Kumar, R & Rogowski, J 2008, 'Neighborhoods and obesity in later life', *American Journal of Public Health,* vol. 98, no. 11, pp. 2065-2071.

Granovetter, M 1978, 'Threshold models of collective behavior', *American Journal of Sociology,* vol. 83, no. 6, pp. 1420-1443.

Grant, J, Chittleborough, C, Taylor, A, Dal Grande, E, Wilson, D, Phillips, P, Adams, R, Cheek, J, et al. 2006, 'The North West Adelaide Health Study: detailed methods and baseline segmentation of a cohort for chronic diseases', *Epidemiological Perspectives and Innovations,* vol. 3, no. 4, DOI:10.1186/1742-5573-3-4.

Grant, J, Taylor, A, Ruffin, R, Wilson, D, Phillips, P, Adams, R & Price, K 2009, 'Cohort profile: The North West Adelaide Health Study (NWAHS)', *International Journal of Epidemiology,* vol. 38, pp. 1479-1486.

Grasser, G, Van Dyck, D, Titze, S & Stronegger, W 2013, 'Objectively measured walkability and active transport and weight-related outcomes in adults: a systematic review', *International Journal of Public Health,* vol. 58, no. 4, pp. 615-625.

Greaves, CJ, Sheppard, KE, Abraham, C, Hardeman, W, Roden, M, Evans, PH & Schwarz, P 2011, 'Systematic review of reviews of intervention components associated with increased effectiveness in dietary and physical activity interventions', *BMC Public Health,* vol. 11, no. 1, p. 119.

Greaves, DR & Channon, KM 2002, 'Inflammation and immune responses in atherosclerosis', *Trends in Immunology,* vol. 23, no. 11, pp. 535-541.

Green, LW & Kreuter, MW 2005, *Health Program Planning: an Educational and Ecological Approach*, 4 edn, McGraw-Hill, Boston.

Greiner, M, Pfeiffer, D & Smith, RD 2000, 'Principles and practical application of the receiver-operating characteristic analysis for diagnostic tests', *Preventive Veterinary Medicine,* vol. 45, no. 1–2, pp. 23-41.

Grøntved, A & Hu, FB 2011, 'Television viewing and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a meta-analysis', *Journal of the American Medical Association,* vol. 305, no. 23, pp. 2448-2455.

Grundy, SM, Pasternak, R, Greenland, P, Smith, S & Fuster, V 1999, 'Assessment of cardiovascular risk by use of multiple-risk-factor assessment equations : a statement for healthcare professionals from the American Heart Association and the American College of Cardiology', *Circulation,* vol. 100, no. 13, pp. 1481-1492.

Hajna, S, Ross, NA, Brazeau, A-S, Bélisle, P, Joseph, L & Dasgupta, K 2015, 'Associations between neighbourhood walkability and daily steps in adults: a systematic review and meta-analysis', *BMC Public Health,* vol. 15, no. 1, pp. 1-8.

Hammond, D, Fong, GT, Zanna, MP, Thrasher, JF & Borland, R 2006, 'Tobacco denormalization and industry beliefs among smokers from four countries', *American Journal of Preventive Medicine,* vol. 31, no. 3, pp. 225-232.

Hammond, RA 2009, 'Complex systems modeling for obesity research', *Preventing Chronic Disease,* vol. 6, no. 3, DOI: http://www.cdc.gov/pcd/issues/2009/jul/09_0017.htm.

Hammond, RA & Ornstein, JT 2014, 'A model of social influence on body mass index', *Annals of the New York Academy of Sciences,* vol. 1331, pp. 34-42.

Harvey, D 2010, *Social Justice and the City*, University of Georgia Press, Athens, Georgia.

He, FJ, Nowson, CA & MacGregor, GA 2006, 'Fruit and vegetable consumption and stroke: meta-analysis of cohort studies', *The Lancet,* vol. 367, no. 9507, pp. 320-326.

He, K, Hu, FB, Colditz, GA, Manson, JE, Willett, WC & Liu, S 2004, 'Changes in intake of fruits and vegetables in relation to risk of obesity and weight gain among middle-aged women', *International Journal of Obesity,* vol. 28, no. 12, pp. 1569-1574.

Healy, GN, Dunstan, DW, Salmon, J, Cerin, E, Shaw, JE, Zimmet, PZ & Owen, N 2007, 'Objectively measured light-intensity physical activity is independently associated with 2-h plasma glucose', *Diabetes Care,* vol. 30, no. 6, pp. 1384-1389.

Heidenreich, PA, Trogdon, JG, Khavjou, OA, Butler, J, Dracup, K, Ezekowitz, MD, Finkelstein, EA, Hong, Y, et al. 2011, 'Forecasting the future of cardiovascular disease in the United States', *Circulation,* vol. 123, no. 8, p. 933.

Hershey, JC, Niederdeppe, J, Evans, WD, Nonnemaker, J, Blahut, S, Holden, D, Messeri, P & Haviland, ML 2005, 'The theory of "truth": how counterindustry campaigns affect smoking behavior among teens', *Health Psychology,* vol. 24, no. 1, pp. 22-31.

Hertzman, C & Boyce, T 2010, 'How experience gets under the skin to create gradients in developmental health', *Annual Review of Public Health,* vol. 31, pp. 329-347

Hill, AB 1965, 'The environment and disease: association or causation?', *Proceedings of the Royal Society of Medicine,* vol. 58, no. 5, pp. 295-300.

Hillsdon, M, Panter, J, Foster, C & Jones, A 2006, 'The relationship between access and quality of urban green space with population physical activity', *Public Health,* vol. 120, no. 12, pp. 1127-1132.

Hippisley-Cox, J, Coupland, C, Vinogradova, Y, Robson, J, May, M & Brindle, P 2007, 'Derivation and validation of QRISK, a new cardiovascular disease risk score for the United Kingdom: prospective open cohort study', *British Medical Journal,* vol. 335, no. 7611, pp. 136-148.

Holsten, JE 2009, 'Obesity and the community food environment: a systematic review', *Public Health Nutrition,* vol. 12, no. 3, pp. 397-405.

Hu, FB, Li, TY, Colditz, GA, Willett, WC & Manson, JE 2003, 'Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women', *Journal of the American Medical Association,* vol. 289, no. 14, pp. 1785-1791.

Humpel, N, Owen, N & Leslie, E 2002, 'Environmental factors associated with adults' participation in physical activity: a review', *American Journal of Preventive Medicine,* vol. 22, no. 3, pp. 188-199.

Hunter, RF, Christian, H, Veitch, J, Astell-Burt, T, Hipp, JA & Schipperijn, J 2015, 'The impact of interventions to promote physical activity in urban green space: a systematic review and recommendations for future research', *Social Science and Medicine,* vol. 124, pp. 246-256.

IDF 2006, *The IDF consensus worldwide definition of the metabolic syndrome*, International Diabetes Federation, Brussels. <http://www.idf.org/webdata/docs/MetS_def_update2006.pdf>.

IDF 2011, *IDF Diabetes Atlas*, 5th edn, International Diabetes Federation, Brussels.

IDF 2013, *IDF Diabetes Atlas*, 6th edn, International Diabetes Federation, Brussels.

IDF 2015, *IDF Diabetes Atlas*, 7 edn, International Diabetes Federation, Brussels.

IEC 2009, 'International Expert Committee Report on the role of the A1C assay in the diagnosis of diabetes', *Diabetes Care,* vol. 32, no. 7, pp. 1327-1334.

Isomaa, B, Almgren, P, Tuomi, T, Forsén, B, Lahti, K, Nissén, M, Taskinen, M-R & Groop, L 2001, 'Cardiovascular morbidity and mortality associated with the metabolic syndrome', *Diabetes Care,* vol. 24, no. 4, pp. 683-689.

Jackson, R, Lawes, CMM, Bennett, DA, Milne, RJ & Rodgers, A 2005, 'Treatment with drugs to lower blood pressure and blood cholesterol based on an individual's absolute cardiovascular risk', *The Lancet,* vol. 365, no. 9457, pp. 434-441.

Jacobson, RP, Mortensen, CR & Cialdini, RB 2011, 'Bodies obliged and unbound: differentiated response tendencies for injunctive and descriptive social norms', *Journal of Personality and Social Psychology,* vol. 100, no. 3, pp. 433-448.

Jakes, RW, Day, NE, Khaw, K-T, Luben, R, Oakes, S, Welch, A, Bingham, S & Wareham, NJ 2003, 'Television viewing and low participation in vigorous recreation are independently associated with obesity and markers of cardiovascular disease risk: EPIC-Norfolk population-based study', *European Journal of Clinical Nutritrion,* vol. 57, no. 9, pp. 1089-1096.

Janiszewski, PM & Ross, R 2007, 'Physical activity in the treatment of obesity: beyond body weight reduction', *Applied Physiology, Nutrition & Metabolism,* vol. 32, no. 3, pp. 512-522.

Jelinski, DE & Wu, J 1996, 'The modifiable areal unit problem and implications for landscape ecology', *Landscape Ecology,* vol. 11, no. 3, pp. 129-140.

Jenkins, DA, Wong, JW, Kendall, CC & et al. 2009, 'The effect of a plant-based low-carbohydrate ("eco-atkins") diet on body weight and blood lipid concentrations in hyperlipidemic subjects', *Archives of Internal Medicine,* vol. 169, no. 11, pp. 1046-1054.

Johnson, JL, Slentz, CA, Houmard, JA, Samsa, GP, Duscha, BD, Aiken, LB, McCartney, JS, Tanner, CJ & Kraus, WE 2007, 'Exercise training amount and intensity effects on metabolic syndrome (from Studies of a Targeted Risk Reduction Intervention through Defined Exercise)', *The American Journal of Cardiology,* vol. 100, no. 12, pp. 1759-1766.

Jones, A, Hillsdon, M & Coombes, E 2009, 'Greenspace access, use, and physical activity: understanding the effects of area deprivation', *Preventive Medicine,* vol. 49, no. 6, pp. 500-505.

Kaczynski, AT & Henderson, KA 2007, 'Environmental correlates of physical activity: a review of evidence about parks and recreation', *Leisure Sciences,* vol. 29, no. 4, pp. 315-354.

Kaczynski, AT & Henderson, KA 2008, 'Parks and recreation settings and active living: a review of associations with physical activity function and intensity', *Journal of Physical Activity and Health,* vol. 5, no. 4, pp. 619-632.

Kaczynski, AT, Potwarka, LR, Smale, BJA & Havitz, ME 2009, 'Association of parkland proximity with neighborhood and park-based physical activity: variations by gender and age', *Leisure Sciences,* vol. 31, no. 2, pp. 174-191.

Kahn, R, Buse, J, Ferrannini, E & Stern, M 2005, 'The metabolic syndrome: time for a critical appraisal', *Diabetes Care,* vol. 28, no. 9, pp. 2289-2304.

Kallgren, CA, Reno, RR & Cialdini, RB 2000, 'A focus theory of normative conduct: when norms do and do not affect behavior', *Personality and Social Psychology Bulletin,* vol. 26, no. 8, pp. 1002-1012.

Katz, LF, Kling, JR & Liebman, JB 2001, 'Moving to Opportunity in Boston: early results of a randomized mobility experiment', *The Quarterly Journal of Economics,* vol. 116, no. 2, pp. 607-654.

Kaufman, JS, Kaufman, S & Poole, C 2003, 'Causal inference from randomized trials in social epidemiology', *Social Science & Medicine,* vol. 57, no. 12, pp. 2397-2409.

Kavanagh, AM, Goller, JL, King, T, Jolley, D, Crawford, D & Turrell, G 2005, 'Urban area disadvantage and physical activity: a multilevel study in Melbourne, Australia', *Journal of Epidemiology and Community Health,* vol. 59, no. 11, pp. 934-940.

Kavanagh, K, Jones, KL, Sawyer, J, Kelley, K, Carr, JJ, Wagner, JD & Rudel, LL 2007, 'Trans fat diet induces abdominal obesity and changes in insulin sensitivity in monkeys', *Obesity,* vol. 15, no. 7, pp. 1675-1684.

Kestens, Y, Lebel, A, Daniel, M, Thériault, M & Pampalon, R 2010, 'Using experienced activity spaces to measure foodscape exposure', *Health and Place,* vol. 16, no. 6, pp. 1094-1103.

Khaw, K-T, Wareham, N, Bingham, S, Luben, R, Welch, A & Day, N 2004, 'Association of hemoglobin A1c with cardiovascular disease and mortality in adults: the European Prospective Investigation into Cancer in Norfolk', *Annals of Internal Medicine,* vol. 141, no. 6, pp. 413-420.

King, AC, Sallis, JF, Frank, LD, Saelens, BE, Cain, K, Conway, TL, Chapman, JE, Ahn, DK & Kerr, J 2011, 'Aging in neighborhoods differing in walkability and income: associations with physical activity and obesity in older adults', *Social Science and Medicine,* vol. 73, no. 10, pp. 1525-1533.

Kirkpatrick, SI, Reedy, J, Butler, EN, Dodd, KW, Subar, AF, Thompson, FE & McKinnon, RA 2014, 'Dietary assessment in food environment research: a systematic review', *American Journal of Preventive Medicine,* vol. 46, no. 1, pp. 94-102.

Koohsari, MJ, Badland, H & Giles-Corti, B 2013, '(Re)Designing the built environment to support physical activity: bringing public health back into urban design and planning', *Cities,* vol. 35, pp. 294-298.

Koohsari, MJ, Mavoa, S, Villanueva, K, Sugiyama, T, Badland, H, Kaczynski, AT, Owen, N & Giles-Corti, B 2015a, 'Public open space, physical activity, urban design and public health: concepts, methods and research agenda', *Health and Place,* vol. 33, pp. 75-82.

Koohsari, MJ, Sugiyama, T, Sahlqvist, S, Mavoa, S, Hadgraft, N & Owen, N 2015b, 'Neighborhood environmental attributes and adults' sedentary behaviors: review and research agenda', *Preventive Medicine,* vol. 77, pp. 141-149.

Kormos, C, Gifford, R & Brown, E 2015, 'The influence of descriptive social norm information on sustainable transportation behavior: a field experiment', *Environment and Behavior,* vol. 47, no. 5, pp. 479-501.

Krebs, DL 1970, 'Altruism: an examination of the concept and a review of the literature', *Psychological Bulletin,* vol. 73, no. 4, pp. 258-302.

Lachowycz, K & Jones, AP 2011, 'Greenspace and obesity: a systematic review of the evidence', *Obesity Reviews,* vol. 12, no. 5, pp. e183-e189.

Lakka, H-M, Laaksonen, DE, Lakka, TA, Niskanen, LK, Kumpusalo, E, Tuomilehto, J & Salonen, JT 2002, 'The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men', *Journal of the American Medical Association,* vol. 288, no. 21, pp. 2709-2716.

Lapinski, MK & Rimal, RN 2005, 'An explication of social norms', *Communication Theory,* vol. 15, no. 2, pp. 127-147.

Laraia, BA, Karter, AJ, Warton, EM, Schillinger, D, Moffet, HH & Adler, N 2012, 'Place matters: neighborhood deprivation and cardiometabolic risk factors in the Diabetes Study of Northern California (DISTANCE)', *Social Science and Medicine,* vol. 74, no. 7, pp. 1082-1090.

Larson, NI, Story, MT & Nelson, MC 2009, 'Neighborhood environments: disparities in access to healthy foods in the U.S.', *American Journal of Preventive Medicine,* vol. 36, no. 1, pp. 74-81.

Lau, DCW, Douketis, JD, Morrison, KM, Hramiak, IM, Sharma, AM, Ur, E & for members of the Obesity Canada Clinical Practice Guidelines Expert Panel 2007, '2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary]', *Canadian Medical Association Journal,* vol. 176, no. 8, pp. s1-13.

Lê-Scherban, F, Albrecht, SS, Osypuk, TL, Sánchez, BN & Diez Roux, AV 2014, 'Neighborhood ethnic composition, spatial assimilation, and change in body mass index over time among Hispanic and Chinese immigrants: Multi-Ethnic Study of Atherosclerosis', *American Journal of Public Health,* vol. 104, no. 11, pp. 2138-2146.

Leal, C & Chaix, B 2011, 'The influence of geographic life environments on cardiometabolic risk factors: a systematic review, a methodological assessment and a research agenda', *Obesity Reviews,* vol. 12, no. 3, pp. 217-230.

Leiter, LA, Fitchett, DH, Gilbert, RE, Gupta, M, Mancini, GBJ, McFarlane, PA, Ross, R, Teoh, H, et al. 2011, 'Cardiometabolic risk in Canada: a detailed analysis and position paper by the Cardiometabolic Risk Working Group', *Canadian Journal of Cardiology,* vol. 27, no. 2, DOI:10.1016/j.cjca.2010.12.054.

Leroux, J, Moore, S, Richard, L & Gauvin, L 2012, 'Physical inactivity mediates the association between the perceived exercising behavior of social network members and obesity: a cross-sectional study.', *PLoS ONE,* vol. 7, no. 10, DOI:10.1371/journal.pone.0046558.

Leslie, E, Coffee, N, Frank, L, Owen, N, Bauman, A & Hugo, G 2007, 'Walkability of local communities: using geographic information systems to objectively assess relevant environmental attributes', *Health and Place,* vol. 13, no. 1, pp. 111-122.

Li, F, Harmer, P, Cardinal, BJ & Vongjaturapat, N 2009, 'Built environment and changes in blood pressure in middle aged and older adults', *Preventive Medicine,* vol. 48, no. 3, pp. 237-241.

Libby, P 2001, 'Current concepts of the pathogenesis of the acute coronary syndromes', *Circulation,* vol. 104, no. 3, pp. 365-372.

Liefbroer, AC & Billari, FC 2010, 'Bringing norms back in: a theoretical and empirical discussion of their importance for understanding demographic behaviour', *Population, Space and Place,* vol. 16, no. 4, pp. 287-305.

Lovasi, GS, Hutson, MA, Guerra, M & Neckerman, KM 2009, 'Built environments and obesity in disadvantaged populations', *Epidemiologic Reviews,* vol. 31, pp. 7-20.

Lovasi, GS, Grady, S & Rundle, A 2012, 'Steps forward: review and recommendations for research on walkability, physical activity and cardiovascular health', *Public health reviews,* vol. 33, no. 4, pp. 484-506.

Maas, J, Verheij, RA, Spreeuwenberg, P & Groenewegen, PP 2008, 'Physical activity as a possible mechanism behind the relationship between green space and health: a multilevel analysis', *BMC Public Health,* vol. 8, no. 1, pp. 1-13.

Macintyre, S, Ellaway, A & Cummins, S 2002, 'Place effects on health: how can we conceptualise, operationalise and measure them?', *Social Science and Medicine,* vol. 55, no. 1, pp. 125-139.

Mackenbach, J, Rutter, H, Compernolle, S, Glonti, K, Oppert, J-M, Charreire, H, De Bourdeaudhuij, I, Brug, J, et al. 2014, 'Obesogenic environments: a systematic review of the association between the physical environment and adult weight status, the SPOTLIGHT project', *BMC Public Health,* vol. 14, no. 1, p. 233.

Malambo, P, Kengne, AP, De Villiers, A, Lambert, EV & Puoane, T 2016, 'Built environment, selected risk factors and major cardiovascular disease outcomes: a systematic review', *PLoS ONE,* vol. 11, no. 11, DOI: 10.1371/journal.pone.0166846.

Malik, VS, Schulze, MB & Hu, FB 2006, 'Intake of sugar-sweetened beverages and weight gain: a systematic review', *The American Journal of Clinical Nutrition,* vol. 84, no. 2, pp. 274-288.

Malik, VS, Popkin, BM, Bray, GA, Després, J-P & Hu, FB 2010a, 'Sugar-sweetened beverages, obesity, type 2 diabetes mellitus, and cardiovascular disease risk', *Circulation,* vol. 121, no. 11, pp. 1356-1364.

Malik, VS, Popkin, BM, Bray, GA, Després, J-P, Willett, WC & Hu, FB 2010b, 'Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis', *Diabetes Care,* vol. 33, no. 11, pp. 2477-2483.

Marini, MM 1984, 'Age and sequencing norms in the transition to adulthood', *Social Forces,* vol. 63, no. 1, pp. 229-244.

Markus, HR & Kitayama, S 1991, 'Culture and the self: implications for cognition, emotion, and motivation', *Psychological Review,* vol. 98, no. 2, pp. 224-253.

Marshall, BDL & Galea, S 2015, 'Formalizing the role of agent-based modeling in causal inference and epidemiology', *American Journal of Epidemiology,* vol. 181, no. 2, pp. 92-99.

McCall, DO, McGartland, CP, McKinley, MC, Patterson, CC, Sharpe, P, McCance, DR, Young, IS & Woodside, JV 2009, 'Dietary intake of fruits and vegetables improves microvascular function in hypertensive subjects in a dose-dependent manner', *Circulation,* vol. 119, no. 16, pp. 2153-2160.

McCormack, GR & Shiell, A 2011, 'In search of causality: a systematic review of the relationship between the built environment and physical activity among adults', *The International Journal of Behavioral Nutrition and Physical Activity,* vol. 8, pp. 125-125.

McGann, S, Jancey, J & Tye, M 2013, 'Taking the stairs instead: the impact of workplace design standards on health promotion strategies', *Australasian Medical Journal,* vol. 6, no. 2, pp. 23-28.

McLeroy, K, Bibeau, D, Steckler, A & Glanz, K 1988, 'An ecological perspective on health promotion programs', *Health Education Quarterly,* vol. 15, no. 4, pp. 351-377.

McNeill, AM, Rosamond, WD, Girman, CJ, Golden, SH, Schmidt, MI, East, HE, Ballantyne, CM & Heiss, G 2005, 'The metabolic syndrome and 11-year risk of incident cardiovascular disease in the Atherosclerosis Risk in Communities Study', *Diabetes Care,* vol. 28, no. 2, pp. 385-390.

Mendis, S & Alwan, A 2011, *Prioritized research agenda for prevention and control of noncommunicable diseases*, World Health Organization, Geneva.

Mendis, S, Puska, P & Norrving, B 2011, *Global Atlas on Cardiovascular Disease Prevention and Control*, World Health Organization, Geneva

Mente, A, de Koning, L, Shannon, HS & Anand, SS 2009, 'A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease', *Archives of Internal Medicine,* vol. 169, no. 7, pp. 659-669.

Miller, TM, Abdel-Maksoud, MF, Crane, LA, Marcus, AC & Byers, TE 2008, 'Effects of social approval bias on self-reported fruit and vegetable consumption: a randomized controlled trial', *Nutrition Journal,* vol. 7, no. 1, p. 18.

Mitchell, R & Popham, F 2008, 'Effect of exposure to natural environment on health inequalities: an observational population study', *The Lancet,* vol. 372, no. 9650, pp. 1655-1660.

Mobley, LR, Root, ED, Finkelstein, EA, Khavjou, O, Farris, RP & Will, JC 2006, 'Environment, obesity, and cardiovascular disease risk in low-income women', *American Journal of Preventive Medicine,* vol. 30, no. 4, pp. 327-332.

Montano, DE & Kasprzyk, D 2008, 'Theory of reasoned action, theory of planned behaviour, and the integrated behavioral model', in K Glanz, B Rimer & K Viswanath (eds), *Health Behaviour and Health Education: theory, research and practice*, 4th edn, Jossey-Bass, San Francisco, CA, pp. 67 - 96.

Morland, K, Diez Roux, AV & Wing, S 2006, 'Supermarkets, other food stores, and obesity: the Atherosclerosis Risk in Communities study', *American Journal of Preventive Medicine,* vol. 30, no. 4, pp. 333-339.

Mozaffarian, D, Aro, A & Willett, WC 2009, 'Health effects of trans-fatty acids: experimental and observational evidence', *European Journal of Clinical Nutrition,* vol. 63, no. S2, pp. S5-S21.

Mozaffarian, D, Appel, LJ & Van Horn, L 2011, 'Components of a cardioprotective diet: new insights', *Circulation,* vol. 123, no. 24, pp. 2870-2891.

Mujahid, MS, Diez Roux, AV, Morenoff, JD & Raghunathan, T 2007, 'Assessing the measurement properties of neighborhood scales: from psychometrics to ecometrics', *American Journal of Epidemiology,* vol. 165, no. 8, pp. 858-867.

Mujahid, MS, Diez Roux, AV, Morenoff, JD, Raghunathan, TE, Cooper, RS, Ni, H & Shea, S 2008, 'Neighborhood characteristics and hypertension', *Epidemiology,* vol. 19, no. 4, pp. 590-598.

Murray, GD & Findlay, JG 1988, 'Correcting for the bias caused by drop-outs in hypertension trials', *Statistics in Medicine,* vol. 7, no. 9, pp. 941-946.

Nathan, DM, Turgeon, H & Regan, S 2007, 'Relationship between glycated haemoglobin levels and mean glucose levels over time', *Diabetologia,* vol. 50, no. 11, pp. 2239-2244.

Nathan, DM, McGee, P, Steffes, MW, Lachin, JM & DCCT/EDIC research group 2013, 'Relationship of glycated albumin to blood glucose and glycated hemoglobin (HbA1C) values and to retinopathy, nephropathy and cardiovascular outcomes in the DCCT/EDIC study', *Diabetes,* vol. 63, pp. 282-290.

National Health and Medical Research Council 2013, *Australian Dietary Guidelines*, National Health and Medical Research Council, Canberra.

Ness, AR & Powles, JW 1997, 'Fruit and vegetables, and cardiovascular disease: a review', *International Journal of Epidemiology,* vol. 26, no. 1, pp. 1-13.

Neter, JE, Stam, BE, Kok, FJ, Grobbee, DE & Geleijnse, JM 2003, 'Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials', *Hypertension,* vol. 42, no. 5, pp. 878-884.

Ngo, A, Paquet, C, Howard, N, Coffee, N, Taylor, A, Adams, R & Daniel, M 2014, 'Area-level socioeconomic characteristics, prevalence and trajectories of cardiometabolic risk', *International Journal of Environmental Research and Public Health,* vol. 11, no. 1, pp. 830-848.

Nicoll, G 2007, 'Spatial measures associated with stair use', *American Journal of Health Promotion,* vol. 21, no. 4S, pp. 346-352.

Nicoll, G & Zimring, C 2009, 'Effect of innovative building design on physical activity', *Journal of Public Health Policy,* vol. 30, no. 1, pp. S111-S123.

Nigbur, D, Lyons, E & Uzzell, D 2010, 'Attitudes, norms, identity and environmental behaviour: using an expanded theory of planned behaviour to predict participation in a kerbside recycling programme', *British Journal of Social Psychology,* vol. 49, no. 2, pp. 259-284.

Nolan, JM, Schultz, PW, Cialdini, RB, Goldstein, NJ & Griskevicius, V 2008, 'Normative social influence is underdetected', *Personality and Social Psychology Bulletin,* vol. 34, no. 7, pp. 913-923.

Oakes, JM, Mậsse, LC & Messer, LC 2009, 'Work Group III: methodologic issues in research on the food and physical activity environments: addressing data complexity', *American Journal of Preventive Medicine,* vol. 36, no. 4S, pp. S177-S181.

OECD 2015a, *Cardiovascular Disease and Diabetes: Policies for Better Health and Quality of Care*, OECD Publishing, Paris.

OECD 2015b, *Health at a Glance 2015*, OECD Publishing, Paris.

Okosun, IS, Lyn, R, Davis-Smith, M, Eriksen, M & Seale, P 2010, 'Validity of a continuous metabolic risk score as an index for modeling metabolic syndrome in adolescents', *Ann Epidemiol,* vol. 20, no. 11, pp. 843-851.

Olander, EK & Eves, FF 2011, 'Elevator availability and its impact on stair use in a workplace', *Journal of Environmental Psychology,* vol. 31, no. 2, pp. 200-206.

Openshaw, S & Taylor, PJ 1979, 'A million or so correlation coefficients: three experiments on the modifiable areal unit problem', *Statistical applications in the spatial sciences,* vol. 21, pp. 127-144.

Openshaw, S 1984, *The Modifiable Areal Unit Problem (Concepts and Techniques in Modern Geography)*, GeoBooks, Norwich.

Owen, N, Cerin, E, Leslie, E, duToit, L, Coffee, N, Frank, LD, Bauman, AE, Hugo, G, et al. 2007, 'Neighborhood walkability and the walking behavior of Australian adults', *American Journal of Preventive Medicine,* vol. 33, no. 5, pp. 387-395.

Paeratakul, S, Ferdinand, DP, Champagne, CM, Ryan, DH & Bray, GA 2003, 'Fast-food consumption among US adults and children: dietary and nutrient intake profile', *Journal of the American Dietetic Association,* vol. 103, no. 10, pp. 1332-1338.

Paquet, C, Dube, L, Gauvin, L, Kestens, Y & Daniel, M 2010, 'Sense of mastery and metabolic risk: moderating role of the local fast-food environment', *Psychosocial Medicine,* vol. 72, pp. 324-331.

Paquet, C, Orschulok, TP, Coffee, NT, Howard, NJ, Hugo, G, Taylor, AW, Adams, RJ & Daniel, M 2013, 'Are accessibility and characteristics of public open spaces associated with a better cardiometabolic health?', *Landscape and Urban Planning,* vol. 118, pp. 70-78.

Paquet, C, Coffee, NT, Haren, MT, Howard, NJ, Adams, RJ, Taylor, AW & Daniel, M 2014, 'Food environment, walkability, and public open spaces are associated with incident development of cardio-metabolic risk factors in a biomedical cohort', *Health and Place,* vol. 28, pp. 173-176.

Payne, CR, Niculescu, M, Just, DR & Kelly, MP 2015, 'Shopper marketing nutrition interventions: social norms on grocery carts increase produce spending without increasing shopper budgets', *Preventive Medicine Reports,* vol. 2, pp. 287-291.

Pepe, MS, Janes, H, Longton, G, Leisenring, W & Newcomb, P 2004, 'Limitations of the odds ratio in gauging the performance of a diagnostic, prognostic, or screening marker', *Am J Epidemiol,* vol. 159, no. 9, pp. 882-890.

Pepitone, A 1976, 'Toward a normative and comparative biocultural social psychology', *Journal of Personality and Social Psychology,* vol. 34, no. 4, pp. 641-653.

Pereira, MA, Kartashov, AI, Ebbeling, CB, Van Horn, L, Slattery, ML, Jacobs Jr, DR & Ludwig, DS 2005, 'Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis', *The Lancet,* vol. 365, no. 9453, pp. 36-42.

Perk, J, De Backer, G, Gohlke, H, Graham, I, Reiner, Ž, Verschuren, M, Albus, C, Benlian, P, et al. 2012, 'European Guidelines on cardiovascular disease prevention in clinical practice (version 2012): The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts)', *European Heart Journal,* vol. 33, no. 13, pp. 1635-1701.

Perseghin, G, Price, TB, Petersen, KF, Roden, M, Cline, GW, Gerow, K, Rothman, DL & Shulman, GI 1996, 'Increased glucose transport–phosphorylation and muscle glycogen synthesis after exercise training in insulin-resistant subjects', *New England Journal of Medicine,* vol. 335, no. 18, pp. 1357-1362.

Pikora, T, Giles-Corti, B, Bull, F, Jamrozik, K & Donovan, R 2003, 'Developing a framework for assessment of the environmental determinants of walking and cycling', *Social Science and Medicine,* vol. 56, no. 8, pp. 1693-1703.

Pitney Bowes Business Insight 2007, *Adelaide 2007 StreetPro and Roads*.

Prentice, AM & Jebb, SA 2003, 'Fast foods, energy density and obesity: a possible mechanistic link', *Obesity Reviews,* vol. 4, no. 4, pp. 187-194.

Prince, SA, Adamo, KB, Hamel, ME, Hardt, J, Gorber, SC & Tremblay, M 2008, 'A comparison of direct versus self-report measures for assessing physical activity in adults: a systematic review', *International Journal of Behavioral Nutrition and Physical Activity,* vol. 5, no. 1, p. 56.

Prochaska, JO & Velicer, WF 1997, 'The Transtheoretical Model of health behavior change', *American Journal of Health Promotion,* vol. 12, no. 1, pp. 38-48.

PROS 2012, *South Australian Monitoring and Surveillance System (SAMSS)*, Population Research and Outcome Studies, Adelaide, viewed 27/6/2012, <http://health.adelaide.edu.au/pros/data/samss/>.

Ragland, DR 1992, 'Dichotomizing continuous outcome variables: dependence of the magnitude of association and statistical power on the cutpoint', *Epidemiology,* vol. 3, no. 5, pp. 434-440.

Rahmanian, E, Gasevic, D, Vukmirovich, I & Lear, SA 2014, 'The association between the built environment and dietary intake - a systematic review', *Asia Pacific Journal of Clinical Nutrition,* vol. 23, no. 2, pp. 183-196.

Reilly, RE, Doyle, J, Bretherton, D, Rowley, KG, Harvey, JL, Briggs, P, Charles, S, Calleja, J, et al. 2008, 'Identifying psychosocial mediators of health amongst Indigenous Australians for the Heart Health Project', *Ethnicity and Health,* vol. 13, no. 4, pp. 351-373.

Reno, RR, Cialdini, RB & Kallgren, CA 1993, 'The transsituational influence of social norms', *Journal of Personality and Social Psychology,* vol. 64, no. 1, pp. 104-112.

Riccardi, G & Rivellese, A 2000, 'Dietary treatment of the metabolic syndrome - the optimal diet', *British Journal of Nutrition,* vol. 83, no. S1, pp. s143-148.

Richardson, EA & Mitchell, R 2010, 'Gender differences in relationships between urban green space and health in the United Kingdom', *Social Science and Medicine,* vol. 71, no. 3, pp. 568-575.

Rimal, RN & Real, K 2005, 'How behaviors are influenced by perceived norms: a test of the theory of normative social behavior', *Communication Research,* vol. 32, no. 3, pp. 389-414.

Riva, M, Gauvin, L, Apparicio, P & Brodeur, JM 2009, 'Disentangling the relative influence of built and socioeconomic environments on walking: the contribution of areas homogenous along exposures of interest', *Social Science and Medicine,* vol. 69, no. 9, pp. 1296-1305.

Rivis, A & Sheeran, P 2003, 'Descriptive norms as an additional predictor in the theory of planned behaviour: a meta-analysis', *Current Psychology,* vol. 22, no. 3, pp. 218-233.

Robinson, E, Fleming, A & Higgs, S 2014, 'Prompting healthier eating: testing the use of health and social norm based messages', *Health Psychology,* vol. 33, no. 9, pp. 1057-1064.

Robinson, E, Thomas, J, Aveyard, P & Higgs, S 2014, 'What everyone else is eating: a systematic review and meta-analysis of the effect of informational eating norms on eating behavior', *Journal of the Academy of Nutrition and Dietetics,* vol. 114, no. 3, pp. 414-429.

Roeseler, A & Burns, D 2010, 'The quarter that changed the world', *Tobacco Control,* vol. 19, no. S1, pp. i3-i15.

Rogers, EM 2003, *Diffusion of Innovations*, 5th edn, Simon and Schuster, New York.

Rose, G 1985, 'Sick individuals and sick populations', *International Journal of Epidemiology,* vol. 14, no. 1, pp. 32-38.

Rosenheck, R 2008, 'Fast food consumption and increased caloric intake: a systematic review of a trajectory towards weight gain and obesity risk', *Obesity Reviews,* vol. 9, no. 6, pp. 535-547.

Ross, CE & Mirowsky, J 2008, 'Neighborhood socioeconomic status and health: context or composition?', *City & Community,* vol. 7, no. 2, pp. 163-179.

Ross, R, Dagnone, D, Jones, PJH, Smith, H, Paddags, A, Hudson, R & Janssen, I 2000, 'Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men: a randomized controlled trial', *Annals of Internal Medicine,* vol. 133, no. 2, pp. 92-103.

Ross, R, Janssen, I, Dawson, J, Kungl, A-M, Kuk, JL, Wong, SL, Nguyen-Duy, T-B, Lee, S, et al. 2004, 'Exercise-induced reduction in obesity and insulin resistance in women: a randomized controlled trial', *Obesity Research,* vol. 12, no. 5, pp. 789-798.

Saelens, BE, Glanz, K, Sallis, JF & Frank, LD 2007, 'Nutrition Environment Measures Study in Restaurants (NEMS-R): development and evaluation', *American Journal of Preventive Medicine,* vol. 32, no. 4, pp. 273-281.

Saelens, BE & Handy, SL 2008, 'Built environment correlates of walking: a review', *Medicine and Science in Sports and Exercise,* vol. 40, no. 7S, pp. S550-S566.

Sallis, JF, Frank, LD, Saelens, BE & Kraft, MK 2004, 'Active transportation and physical activity: opportunities for collaboration on transportation and public health research', *Transportation Research Part A: Policy and Practice,* vol. 38, no. 4, pp. 249-268.

Sallis, JF, Cervero, RB, Ascher, W, Henderson, KA, Kraft, MK & Kerr, J 2006, 'An ecological approach to creating active living communities', *Annual Review of Public Health,* vol. 27, pp. 297-322.

Sallis, JF, Owen, N & Fisher, EB 2008, 'Ecological models of health behaviour', in K Glanz, B Rimer & K Viswanath (eds), *Health Behaviour and Health Education: Theory, Research and Practice*, 4th edn, Jossey-Bass, San Fransisco, pp. 465 - 485.

Sallis, JF, Saelens, BE, Frank, LD, Conway, TL, Slymen, DJ, Cain, KL, Chapman, JE & Kerr, J 2009, 'Neighborhood built environment and income: examining multiple health outcomes', *Social Science and Medicine,* vol. 68, no. 7, pp. 1285-1293.

Sallis, JF, Floyd, MF, Rodríguez, DA & Saelens, BE 2012, 'Role of built environments in physical activity, obesity, and cardiovascular disease', *Circulation,* vol. 125, no. 5, pp. 729-737.

Schüle, SA & Bolte, G 2015, 'Interactive and independent associations between the socioeconomic and objective built environment on the neighbourhood level and individual health: a systematic review of multilevel studies', *PLoS ONE,* vol. 10, no. 4, DOI: 10.1371/journal.pone.0123456.

Schultz, PW 1999, 'Changing behavior with normative feedback interventions: a field experiment on curbside recycling', *Basic and Applied Social Psychology,* vol. 21, no. 1, pp. 25-36.

Schultz, PW, Nolan, JM, Cialdini, RB, Goldstein, NJ & Griskevicius, V 2007, 'The constructive, destructive, and reconstructive power of social norms', *Psychological Science,* vol. 18, no. 5, pp. 429-434.

Selvin , E, Steffes , MW, Zhu , H, Matsushita , K, Wagenknecht , L, Pankow , J, Coresh , J & Brancati , FL 2010, 'Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults', *New England Journal of Medicine,* vol. 362, no. 9, pp. 800-811.

Shaw, JE & Chisholm, D 2003, 'Epidemiology and prevention of type 2 diabetes and the metabolic syndrome', *Medical Journal of Australia,* vol. 173, pp. 379-383.

Shi, L, Morrison, JA, Wiecha, J, Horton, M & Hayman, LL 2011, 'Healthy lifestyle factors associated with reduced cardiometabolic risk', *British Journal of Nutrition,* vol. 105, no. 5, pp. 747-754.

Shigematsu, R, Sallis, J, Conway, T, Saelens, B, Frank, L, Cain, K, Chapman, J & King, A 2009, 'Age differences in the relation of perceived neighborhood environment to walking', *Medicine and Science in Sports and Exercise,* vol. 41, no. 2, p. 314.

Smith, JR, Louis, WR, Terry, DJ, Greenaway, KH, Clarke, MR & Cheng, X 2012, 'Congruent or conflicted? The impact of injunctive and descriptive norms on environmental intentions', *Journal of Environmental Psychology,* vol. 32, no. 4, pp. 353-361.

South Australian Department of Health 2005, *HOS: Self Reported Prevalence of Obesity in the SA Health Regions*, Population Research and Outcome Studies Unit, South Australian Department of Health, Adelaide.

Spatial Planning Analysis and Research Unit 2008, *Guide to using the 2007 Retail Database Adelaide Statistical Division and nearby large Country Centres*, Planning SA, Government of South Australia, Adelaide.

Stamatakis, E, Hamer, M & Dunstan, DW 2011, 'Screen-based entertainment time, all-cause mortality, and cardiovascular events: population-based study with ongoing mortality and hospital events follow-up', *Journal of the American College of Cardiology,* vol. 57, no. 3, pp. 292-299.

Stender, S, Dyerberg, J & Astrup, A 2007, 'Fast food: unfriendly and unhealthy', *International Journal of Obesity,* vol. 31, no. 6, pp. 887-890.

Sugiyama, T, Neuhaus, M, Cole, R, Giles-Corti, B & Owen, N 2012, 'Destination and route attributes associated with adults' walking: a review', *Medicine and Science in Sports and Exercise,* vol. 44, no. 7, pp. 1275-1286.

Sunday, J, Eyles, J & Upshur, R 2001, 'Applying Aristotle's doctrine of causation to Aboriginal and biomedical understanding of diabetes', *Culture, Medicine and Psychiatry,* vol. 25, no. 1, pp. 63-85.

Sundquist, K, Eriksson, U, Kawakami, N, Skog, L, Ohlsson, H & Arvidsson, D 2011, 'Neighborhood walkability, physical activity, and walking behavior: The Swedish Neighborhood and Physical Activity (SNAP) study', *Social Science and Medicine,* vol. 72, no. 8, pp. 1266-1273.

Sundquist, K, Eriksson, U, Mezuk, B & Ohlsson, H 2015, 'Neighborhood walkability, deprivation and incidence of type 2 diabetes: a population-based study on 512,061 Swedish adults', *Health and Place,* vol. 31, pp. 24-30.

Susser, M 1999, 'Should the epidemiologist be a social scientist or a molecular biologist?', *International Journal of Epidemiology,* vol. 28, no. 5, pp. s1019-1021.

Svendsen, M, Blomhoff, R, Holme, I & Tonstad, S 2007, 'The effect of an increased intake of vegetables and fruit on weight loss, blood pressure and antioxidant defense in subjects with sleep related breathing disorders', *European Journal of Clinical Nutrition,* vol. 61, no. 11, pp. 1301-1311.

Tamosiunas, A, Grazuleviciene, R, Luksiene, D, Dedele, A, Reklaitiene, R, Baceviciene, M, Vencloviene, J, Bernotiene, G, et al. 2014, 'Accessibility and use of urban green spaces, and cardiovascular health: findings from a Kaunas cohort study', *Environmental Health,* vol. 13, no. 1, DOI: 10.1186/1476-069x-13-20.

Taylor, BT, Fernando, P, Bauman, AE, Williamson, A, Craig, JC & Redman, S 2011, 'Measuring the quality of public open space using Google Earth', *American Journal of Preventive Medicine,* vol. 40, no. 2, pp. 105-112.

Thaler, RH, Sunstein, CR & Balz, JP 2013, 'Choice Architecture ', in E Shafir (ed), *The Behavioral Foundations of Public Policy*, Princeton University Press, Princeton.

Thompson, PD, Crouse, SF, Goodpaster, B, Kelley, D, Moyna, N & Pescatello, L 2001, 'The acute versus the chronic response to exercise', *Medicine and Science in Sports and Exercise,* vol. 33, no. 6, pp. s438-445.

Thornton, LE, Crawford, DA & Ball, K 2010, 'Neighbourhood-socioeconomic variation in women's diet: the role of nutrition environments', *European Journal of Clinical Nutrition,* vol. 64, no. 12, pp. 1423-1432.

Tobacco Control Section 1998, *A Model for Change: the California Experience in Tobacco Control*, California Department of Health Services, Sacramento.

Tobler, WR 1970, 'A computer movie simulating urban growth in the Detroit region', *Economic Geography,* vol. 46, pp. 234-240.

Trewin, D 2006, *Census Dictionary*, cat.no. ABS 2901.0, Australian Bureau of Statistics, Commonwealth of Australia, Canberra.

Troped, PJ, Wilson, JS, Matthews, CE, Cromley, EK & Melly, SJ 2010, 'The built environment and location-based physical activity', *American Journal of Preventive Medicine,* vol. 38, no. 4, pp. 429-438.

Tunstall-Pedoe, H 2003, *MONICA, monograph and multimedia sourcebook: world's largest study of heart disease, stroke, risk factors, and population trends 1979-2002*, World Health Organization, Geneva.

Turner, RC & Holman, RR 1995, 'Lessons from UK prospective diabetes study', *Diabetes Research and Clinical Practice,* vol. 28, pp. s151-157.

Turrell, G, Blakely, T, Patterson, C & Oldenburg, B 2004, 'A multilevel analysis of socioeconomic (small area) differences in household food purchasing behaviour', *Journal of Epidemiology and Community Health,* vol. 58, no. 3, pp. 208-215.

Turrell, G & Giskes, K 2008, 'Socioeconomic disadvantage and the purchase of takeaway food: a multilevel analysis', *Appetite,* vol. 51, pp. 69-91.

Turrell, G, Bentley, R, Thomas, LR, Jolley, D, Subramanian, S & Kavanagh, AM 2009, 'A multilevel study of area socio-economic status and food purchasing behaviour', *Public Health Nutrition,* vol. 12, no. 11, pp. 2074–2083.

Unal, B, Critchley, JA & Capewell, S 2004, 'Explaining the decline in coronary heart disease mortality in England and Wales between 1981 and 2000', *Circulation,* vol. 109, no. 9, pp. 1101-1107.

Van Cauwenberg, J, De Bourdeaudhuij, I, De Meester, F, Van Dyck, D, Salmon, J, Clarys, P & Deforche, B 2011, 'Relationship between the physical environment and physical activity in older adults: a systematic review', *Health and Place,* vol. 17, no. 2, pp. 458-469.

van den Berg, M, Wendel-Vos, W, van Poppel, M, Kemper, H, van Mechelen, W & Maas, J 2015, 'Health benefits of green spaces in the living environment: a systematic review of epidemiological studies', *Urban Forestry and Urban Greening,* vol. 14, no. 4, pp. 806-816.

Van Dyck, D, Cerin, E, Cardon, G, Deforche, B, Sallis, JF, Owen, N & de Bourdeaudhuij, I 2010, 'Physical activity as a mediator of the associations between neighborhood walkability and adiposity in Belgian adults', *Health and Place,* vol. 16, no. 5, pp. 952-960.

Vasudevan, AR & Ballantyne, CM 2005, 'Cardiometabolic risk assessment: an approach to the prevention of cardiovascular disease and diabetes mellitus', *Clinical Cornerstone,* vol. 7, no. 2/3, pp. 7-16.

Wannamethee, SG, Shaper, AG, Lennon, L & Morris, RW 2005, 'Metabolic syndrome vs Framingham risk score for prediction of coronary heart disease, stroke, and type 2 diabetes mellitus', *Archives of Internal Medicine,* vol. 165, no. 22, pp. 2644-2650.

Weber, M [1922] 1978, *Economy and Society: an outline of interpretive sociology*, University of California Press, Berkeley.

West, BT, Welch, KB & Galecki, AT 2007, *Linear Mixed Models: A Practical Guide Using Statistical Software*, Chapman & Hall/CRC, Boca Raton.

WHO 1986, *The Ottawa Charter for Health Promotion*, The World Health Organization, Geneva, viewed 15/4/2014, <http://www.who.int/healthpromotion/conferences/previous/ottawa/en/>.

WHO 1999, *Definition, diagnosis and classification of diabetes mellitus and its complications*, World Health Organization, Geneva.

WHO 2006, *Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia: report of a WHO/IDF consultation*, World Health Organisation, Geneva.

WHO 2011a, *Global status report on noncommunicable diseases 2010*, World Health Organization, Geneva.

WHO 2011b, *Use of glycated haemoglobin (HbA_{1c}) in the diagnosis of diabetes mellitus: abbreviated report of a WHO consultation* World Health Organisation, Geneva.

WHO 2013, *Global action plan for the prevention and control of noncommunicable diseases 2013-2020*, World Health Organization, Geneva.

WHO 2014, *Global database on body mass index: an interactive surveillance tool for monitoring nutrition transition*, World Health Organization,, Geneva, viewed 11/9/2014, <http://apps.who.int/bmi/index.jsp>.

WHO 2016, *Global report on diabetes*, World Health Organization, Geneva.

Wijndaele, K, Gaston, B, Duvigneaud, N, Matton, L, Duquet, W, Thomis, M, Lefevre, J & Philippaerts, RM 2006, 'A continuous metabolic syndrome risk score', *Diabetes Care,* vol. 29, no. 10, p. 2329.

Wijndaele, K, Duvigneaud, N, Matton, L, Duquet, W, Delecluse, C, Thomis, M, Beunen, G, Lefevre, J & Philippaerts, RM 2009, 'Sedentary behaviour, physical activity and a continuous metabolic syndrome risk score in adults', *European Journal of Clinical Nutrition,* vol. 63, no. 3, pp. 421-429.

Wilcox, S, Castro, C, King, AC, Housemann, R & Brownson, RC 2000, 'Determinants of leisure time physical activity in rural compared with urban older and ethnically diverse women in the United States', *Journal of Epidemiology and Community Health,* vol. 54, no. 9, pp. 667-672.

Willi, C, Bodenmann, P, Ghali, WA, Faris, PD & Cornuz, J 2007, 'Active smoking and the risk of type 2 diabetes: a systematic review and meta-analysis', *Journal of the American Medical Association,* vol. 298, no. 22, pp. 2654-2664.

Wilson, PWF, Meigs, JB, Sullivan, L, Fox, CS, Nathan, DM & D'Agostino, RB 2007, 'Prediction of incident diabetes mellitus in middle-aged adults: The Framingham Offspring Study', *Archives of Internal Medicine,* vol. 167, no. 10, pp. 1068-1074.

Wilson, PWF & Meigs, JB 2008, 'Cardiometabolic risk: a Framingham perspective', *International Journal of Obesity,* vol. 32, no. S2, pp. S17-20.

Woodward, M, Brindle, P & Tunstall-Pedoe, H 2007, 'Adding social deprivation and family history to cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended Cohort (SHHEC)', *Heart,* vol. 93, pp. 173-176.

Yen, IH & Kaplan, GA 1998, 'Poverty area residence and changes in physical activity level: Evidence from the Alameda County Study', *American Journal of Public Health,* vol. 88, no. 11, pp. 1709-1712.

Yen, IH, Michael, YL & Perdue, L 2009, 'Neighborhood environment in studies of health of older adults: a systematic review', *American Journal of Preventive Medicine,* vol. 37, no. 5, pp. 455-463.

Zenk, SN, Schulz, AJ, Israel, BA, James, SA, Bao, S & Wilson, ML 2005, 'Fruit and vegetable access differs by community racial composition and socioeconomic position in Detroit, Michigan', *Ethnicity and Disease,* vol. 16, no. 1, pp. 275-280.

Zenk, SN, Schulz, AJ, Matthews, SA, Odoms-Young, A, Wilbur, J, Wegrzyn, L, Gibbs, K, Braunschweig, C & Stokes, C 2011, 'Activity space environment and dietary and physical activity behaviors: a pilot study', *Health and Place,* vol. 17, no. 5, pp. 1150-1161.

Appendices

Appendix A: PAMS Ethics Approvals

Government of South Australia
SA Health

Ethics of Human Research Committee

The Queen Elizabeth Hospital
28 Woodville Road
WOODVILLE SOUTH SA 5011
Lyell McEwin Hospital
Haydown Road
ELIZABETH VALE SA 5112

22 February 2010

Professor M Daniel
Research Chair for Social Epidemiology
School of Health Sciences
University of South Australia
GPO BOX 2471
ADELAIDE SA 5001

Dear Professor Daniel Application Number 2010010

The Ethics of Human Research Committee at the last meeting considered your protocol entitled:

"Place and Metabolic Syndrome"

The following documents have been reviewed and approved:

- CNAHS Ethics of Human Research Committee (TQEH & LMH) Application Form

Approval Status: **FINAL**

Period of Approval: **23 February 2010 – 23 February 2011**

***Please note the terms under which Ethical approval is granted:**

1. Researchers are required to immediately report to the Ethics of Human Research Committee anything which might warrant review of ethical approval of the protocol, including:

a) serious or unexpected adverse effects on participants;
b) proposed changes in the protocol; and
c) unforseen events that might affect continued ethical acceptability of the project

2. Protocols are approved for up to twelve months only and a report is required at the end of the study or 12 month period. Extensions will not be granted without a report to the Committee.
3. Confidentiality of the research subjects shall be maintained at all times as required by law
4. All research subjects shall be provided with a Patient Information Sheet and Consent Form, unless otherwise approved by the Committee
5. The Patient Information Sheet and Consent Form shall be printed on the relevant site letterhead stating the contact details for the researchers
6. The Patient Information Sheet must state that the Executive Officer can be contacted for information regarding conduct of the study, policies and procedures, or if the participant wishes to make a confidential complaint
7. A report and a copy of any published material should be forwarded to the Committee at the completion of the project.

Yours sincerely

[redacted]

A/Professor Timothy Mathew
Chairman
Central Northern Adelaide Health Service
Ethics of Human Research Committee (TQEH & LMH)

Ethics of Human Research Committee (TQEH & LMH)
Ph: +61 08 8222 6841

From: Alice Granger
Sent: Wednesday, 24 February 2010 1:03 PM
To: Mark Daniel
Cc: graeme.hugo@adelaide.edu.au; Catherine Paquet; anne.taylor@health.sa.gov.au; robert.adams@adelaide.edu.au; Margaret Cargo
Subject: Ethics Protocol P030/10 "Linking place to metabolic syndrome via behavioural and psychosocial antecedents: levers for public health intervention"
Attachments: Conditions September 2008.doc

Dear Mark,

Re: **Ethics Protocol P030/10 "Linking place to metabolic syndrome via behavioural and psychosocial antecedents: levers for public health intervention"**

Thank you for submitting your ethics protocol for consideration. Your protocol has been considered by the Deputy Chairperson of the University's Human Research Ethics Committee on behalf of the Committee.

I am pleased to advise that your protocol has been approved subject to you:

1. Obtaining ethics approval from the Central Northern Adelaide Health Service Ethics of Human Research Committee
2. Amending section 5.1 as the University now requires researchers to store data for only 5 years
3. Finalising the multi-party agreement for ownership of data (section 5.1).
4. Amending the information sheet to include the following statement "This project has been approved by the University of South Australia's Human Research Ethics Committee. If you have any ethical concerns about the project or questions about your rights as a participant please contact the Executive Officer or this Committee, tel: +61 8 8302 3118; email: vicki.allen@unisa.edu.au"

Please note that the Deputy Chairperson's decision will be reported to the next meeting of the Human Research Ethics Committee for endorsement.

Please regard this email as formal notification of assessment.

Under the national guidelines, you must not begin your research before receiving final approval. Once you have submitted the necessary amendments, and they have been accepted, your approval will be finalised and you may commence your research.

Ethics approval is always made on the basis of a number of conditions detailed in the attachment; it is important that you are familiar with, and abide by, these conditions. It is also essential that you conduct all research according to UniSA guidelines, which can be found at http://www.unisa.edu.au/res/ethics/human.asp. These guidelines should assist you with the formulation of necessary papers such as the information sheet and the consent form.

Please note, if your project is a clinical trial you are required to register it in a publicly accessible trials registry prior to enrolment of the first participant (eg Australian New Zealand Clinical Trials Registry http://www.anzctr.org.au/Survey/UserQuestion.aspx) as a condition of ethics approval.

We look forward to receiving your amendments in the near future. In the meantime, please contact me if you have any questions.

Kind regards

Alice

Alice Granger
Compliance Officer
Research and Innovation Services
University of South Australia
Mawson Lakes Campus
Tel: 08 8302 3523
email: alice.granger@unisa.edu.au

CRICOS provider number 00121B

Government of South Australia
SA Health

Human Research Ethics Committee

ABN 97 643 356 590

Level 10, CitiCentre
11 Hindmarsh Square
Adelaide SA 5000

PO Box 287
Rundle Mall
Adelaide 5000
Telephone (08) 8226 6064
Facsimile (08) 8226 7088

Professor Mark Daniel
Research Chair for Social Epidemiology
School of Health Sciences
University of South Australia
GPO Box 2471
ADELAIDE SA 5001

Dear Professor Daniel,

Re: Linking place to metabolic syndrome via behavioural and psychosocial antecedents: Levers for public health intervention.

HREC PROTOCOL NO: 354/03/2013

Thank you for submitting the above proposal to the SA Health Human Research Ethics Committee for consideration. The Committee reviewed your application at its meeting on 7th April.

I am pleased to advise that ethics approval has been granted to your project, subject to the following conditions:

- The research being conducted in accordance with the 'National Statement on Ethical Conduct in Human Research.'
- Provision of a final report when the project is completed.
- Immediate notification to HREC of any complaints by or adverse events involving participants.
- Immediate notification of any unforeseen events that might affect continued ethical acceptability of the project.
- Submission of any significant changes to the original proposal. Such changes should be approved by the HREC before they are implemented.

- Immediate advice, giving reasons, if the project is discontinued before its completion.

Approval is given for a period of three (3) years only, and if the research is more prolonged than this, a new submission will be required.

Should you have any questions or concerns, please contact Sarah Lawson, Executive Officer of the HREC, Tel 8226 6367 or E-mail hrec@health.sa.gov.au

We wish you well with your project.

Yours sincerely,



David Roder
A/CHAIRPERSON
HUMAN RESEARCH ETHICS COMMITTEE

14/4/2010

Catherine Paquet

From: Alice Granger
Sent: Thursday, 10 June 2010 1:55 PM
To: Mark Daniel
Cc: graeme.hugo@adelaide.edu.au; anne.taylor@health.sa.gov.au; Catherine Paquet; robert.adams@adelaide.edu.au; Maria Zollo
Subject: Ethics protocol P029/10 "Testing the behaviour and psychological mechanisms underlying geographic variation in metabolic syndrome"
Attachments: Conditions September 2008.doc

Follow Up Flag: Follow up
Flag Status: Flagged

Dear Mark,

Re: **Ethics protocol P029/10 "Testing the behaviour and psychological mechanisms underlying geographic variation in metabolic syndrome"**

Thank you for providing the amendments and/or additional information requested by the Deputy Chairperson of the University's Human Research Ethics Committee (email dated 24th February 2010).

I am pleased to advise that your protocol has been granted ethics approval and meets the requirements of the *National Statement on Ethical Conduct in Human Research*.

Please regard this email as formal notification of approval.

Ethics approval is always made on the basis of a number of conditions detailed in the attachment; it is important that you are familiar with, and abide by, these conditions. It is also essential that you conduct all research according to UniSA guidelines, which can be found at http://www.unisa.edu.au/res/ethics/default.asp

Please note, if your project is a clinical trial you are required to register it in a publicly accessible trials registry prior to enrolment of the first participant (eg Australian New Zealand Clinical Trials Registry http://www.anzctr.org.au/Survey/UserQuestion.aspx) as a condition of ethics approval.

Best wishes for your research.

Alice

Alice Granger
Compliance Officer
Research and Innovation Services
University of South Australia
Mawson Lakes Campus
Tel: 08 8302 3523
email: alice.granger@unisa.edu.au

CRICOS provider number 00121B

Appendix B: Ethic Approval for use of SAMSS data

Government of South Australia
SA Health

SA Health Human Research Ethics Committee
Level 10, Citi-Centre Building
11 Hindmarsh Square
ADELAIDE SA 5000
Telephone: (08) 8226 6367
Facsimile: (08) 8226 7088

Ms Suzanne Carroll
Social Epidemiology and Evaluation Research Group
IPC CEA-9
Sansom Institute for Health Research & School of Population Health
University of South Australia
ADELAIDE SA 5001

Dear Ms Carroll,

HREC reference number: HREC/13/SAH/52

Project title: Evaluating the relative and interactive contributions of contextual and compositional characteristics to the evolution of cardiometabolic risk

RE: HREC Application – Approval

Thank you for submitting the above project, which was considered by the SA Health HREC at its meeting held on 1st May 2013.

I am pleased to advise that your application has been granted full ethics approval and appears to meet the requirements of the *National Statement on Ethical Conduct in Human Research*.

Please note the following conditions of approval:

- The research must be conducted in accordance with the 'National Statement on Ethical Conduct in Human Research.'
- A progress report, at least annually, must be provided to the HREC.
- When the project is completed, a final report must be provided to the HREC.
- The HREC must be notified of any complaints by participants or of adverse events involving participants.
- The HREC must be notified immediately of any unforeseen events that might affect ethical acceptability of the project.
- Any proposed changes to the original proposal must be submitted to and approved by the HREC before they are implemented.
- If the project is discontinued before its completion, the HREC must be advised immediately and provided with reasons for discontinuing the project.

HREC approval is valid for 3 years from the date of this letter.

[redacted]

Should you have any queries about the HREC's consideration of your project please contact Lauren Perry, Executive Officer of the HREC, on (08) 8226 6431 or hrec@health.sa.gov.au

You are reminded that this letter constitutes ethical approval only. You must not commence this research project at a SA Health site until separate authorisation from the Chief Executive or delegate of that site has been obtained via the completion of a Site Specific Assessment form. Please contact David van der Hoek via email at ResearchGovernance@health.sa.gov.au to discuss this process further.

The HREC wishes you every success in your research.

Yours sincerely

[redacted]

Andrew Alston
DEPUTY CHAIRPERSON
HUMAN RESEARCH ETHICS COMMITTEE

6/5/13

Appendix C: Postcodes for NWAHS baseline sampling

Baseline sampling for the North West Adelaide Health Study (NWAHS) by Postcode and suburb name, Stage 1 1999-2003

Australia Post® Postcodes that were originally sampled in the north-west population:

5007-5025

5031-5033

5037

5040

5088-5093

5095-5098

5106-5117

5120-5121

5125-5127

Within both Australia Post® Postcodes and suburb for inclusion within baseline sample:

5035

5037

5038

5082

5094

5118

Ashford, Camden Park, Cavan, Dry Creek, Gawler, Gawler East, Gawler South, Gawler West, Glandore, Keswick, Keswick Terminal, Kurralta Park, Netley, North Plympton, Ovingham, Plympton, Plympton North, Willaston

Appendix D: Codes for Retail Database extractions

"Net retail area" used in constructing walkability used all retail codes (not just the provided list)

Table D1: Food outlet data extraction and classifications

Category	Retail Code	Retail Code Description
Fast food outlets		
Major fast-food franchises (i.e., more than 10 franchises)		
Hungry Jacks	117	Take-away
McDonalds	117	Take-away
KFC	117	Take-away
Pizza Hut	117	Take-away
Red Rooster	117	Take-away
Barnacle Bill	117	Take-away
Dominos	117	Take-away
Independent fast-food takeaway stores (i.e., independent store selling high-caloric food)		
Fish and Chips	117	Take-away
Hamburgers, Hot Dogs	117	Take-away
Yiros	117	Take-away
Cooked chicken	117	Take-away
Combination of the above	117	Take-away
Pizza	117	Take-away
Pies, Sausage rolls, Pasties	117	Take-away
Other	117	Take-away
Snack bars	115	Milk Bars
Snack bars	104	Delicatessen
Healthful food resources		
Green grocers	110	Green grocers
Fresh poultry	108	Fresh poultry
Fresh fish	107	Fresh fish
Butcher	102	Butcher
Health foods/nuts/natural remedies	112	Health foods/nuts/natural remedies
Supermarkets (with total floor space > 200m^2, excluding ethnic stores	116	Supermarkets
Supermarkets (large)	803	Supermarkets (large)
Fine food (gourmet, pasta, cheese) classified under grocer	111	Grocer
Fine food (gourmet, pasta, cheese) classified under deli	104	Delicatessen

Appendix E: Framingham Risk Score Algorithms

Framingham General CVD Risk Score Calculator (Tables E1-3) (D'Agostino et al. 2008)

Table E1 Point allocation for Women

Points	Age (years)	HDL	Total Cholesterol	SBP Not Treated	SBP Treated	Smoker	Diabetic	
-3				<120				
-2		60+						
-1		50-59			<120			
0	30-34	45-49	<160	120-129		No	No	
1		35-44	160-199	130-139				
2	35-39	<35		140-149	120-129			
3			200-239		130-139	Yes		
4	40-44		240-279	150-159			Yes	
5	45-49		280+	160+	140-149			
6					150-159			
7	50-54				160+			
8	55-50							
9	60-64							
10	65-69							
11	70-74							
12	75+							
Points allotted								**Total**

SBP indicates systolic blood pressure

Table E2 Point allocation for Men

Points	Age (years)	HDL	Total Cholesterol	SBP Not Treated	SBP Treated	Smoker	Diabetic	
-2		60+		<120				
-1		50-59						
0	30-34	45-49	<160	120-129	<120	No	No	
1		35-44	160-199	130-139				
2	35-39	<35	200-239	140-159	120-129			
3			240-279	160+	130-139		Yes	
4			280+		140-159	Yes		
5	40-44				160+			
6	45-49							
7								
8	50-54							
9								
10	55-59							
11	60-64							
12	65-69							
13								
14	70-74							
15	75+							
Points allotted								Total

SBP indicates systolic blood pressure

Table E3 10-year estimate of CVD risk based on allocated points

Women		Men	
Points	**Risk, %**	**Points**	**Risk, %**
≤ - 2	<1	≤ -3	<1
-1	1.0	-2	1.1
0	1.2	-1	1.4
1	1.5	0	1.6
2	1.7	1	1.9
3	2.0	2	2.3
4	2.4	3	2.8
5	2.8	4	3.3
6	3.3	5	3.9
7	3.9	6	4.7
8	4.5	7	5.6
9	5.3	8	6.7
10	6.3	9	7.9
11	7.3	10	9.4
12	8.6	11	11.2
13	10.0	12	13.2
14	11.7	13	15.6
15	13.7	14	18.4
16	15.9	15	21.6
17	18.5	16	25.3
18	21.5	17	29.4
19	24.8	18+	>30
20	28.5	-	-
21+	>30	-	-

Table E4 Estimated 8-year type 2 diabetes risk for men and women (Wilson et al. 2007)

Items	Item Points	Item Point Total	8-Year Risk of T2DM, %
Fasting glucose level 100-126 mg/dL, yes/no	10	≤10	≤3
BMI 25.0-29.9, yes/no	2	11	4
BMI ≥ 30.0, yes/no	5	12	4
HDL-C level <40 mg/dL in men or <50 mg/dL in women, yes/no	5	13	5
Parental history of diabetes mellitus, yes/no	3	14	6
Triglyceride level ≥ 150 mg/dL, yes/no	3	15	7
Blood pressure ≥ 130/85 mm Hg or receiving treatment, yes/no	2	16	9
Item Point Total		17	11
		18	13
		19	15
		20	18
		21	21
		22	25
		23	29
		24	33
		≥25	>35

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in metres squared); HDL-C, high-density lipoprotein cholesterol; T2DM, type 2 diabetes mellitus

*the unshaded portion of the table shows the possible item point totals and their corresponding 8-year risk of T2DM. After calculating the item point total, check the last 2 columns to determine the risk of T2DM.

Appendix F: Land use codes for 2007 Property Cadastre extraction

Land use code	Description
4110	Vacant allotment conservation or recreation
4210	Wooded area conservation
4500	Reserve
4510	Undeveloped reserve
4520	Developed reserve
5560	Botanical garden and arboretum
7100	Outdoor arenas, sports oval
7105	Outdoor stadium, entertainment area
7110	Athletics
7120	Baseball
7130	Cricket
7140	Football
7141	Australian rules
7142	Soccer
7143	Rugby
7150	Hockey
7160	Lacrosse
7210	Archery
7220	Basketball
7230	Lawn bowls
7240	Croquet
7250	Tennis
7260	Sports ground N.E.C.
7300	Golf course
7310	Golf - pitch and putt
7320	Golf - putt putt
7330	Golf - driving range
7420	Racing track – bicycle
7530	Parks and gardens including picnicking

Adapted from Department for Transport, Energy and Infrastructure 2007, *State Valuation Office*, Department for Transport, Energy and Infrastructure, Government of South Australia, Adelaide.

Appendix G: Authorship statement and copyright for manuscript 1

Carroll, S, Paquet, C, Howard, N, Adams, R, Taylor, A, Daniel, M. 2014, 'Validation of continuous clinical indices of cardiometabolic risk in a cohort of Australian adults', *BMC Cardiovascular Disorders*, vol. 14, no. 1, p. 27, DOI: 10.1186/1471-2261-14-27

Available open access at:

http://bmccardiovascdisord.biomedcentral.com/articles/10.1186/1471-2261-14-27

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Authorship contributions:

Suzanne J. Carroll conceived and designed the study, analysed the data under guidance, contributed to the interpretation of the results, and wrote and approved the manuscript. I certify that the statement of contribution is accurate.

Signed: Date: 24 January 2017

Catherine Paquet conceived and designed the study, provided guidance on the analysis of the study, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

Signed: Date: 25/01/2017

Natasha J. Howard conceived and designed the study, revised the manuscript critically for important intellectual content and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

Signed: Date: 24/01/2017

Robert J. Adams conceived and designed the study and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

Signed: Date: 8/2/17

Anne W. Taylor conceived and designed the study and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

Signed: Date: 24/1/2017

Mark Daniel conceived and designed the study, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

Signed: Date: 24 Jan 2017

Appendix H: Authorship statement and copyright for manuscript 2

Carroll, SJ, Paquet, C, Howard, N, Coffee, NT, Taylor, AW, Niyonsenga, T & Daniel, M, 2016 "Local descriptive norms for overweight/obesity and physical inactivity, features of the built environment, and 10-year change in glycosylated haemoglobin in an Australian population-based biomedical cohort", *Social Science & Medicine*, vol. 166, pp. 233-243.

Permission to reprint article granted through the Copyright Clearance Centre on Nov 28 2016 (Copyright license 3997960215768 – a copy follows the authorship information)

Authorship contributions:

Suzanne J. Carroll conceived and designed the study, made a significant contribution to the acquisition and preparation of data used in the study, analysed the data, contributed to the interpretation of the results, and wrote and approved the manuscript. I certify that the statement of contribution is accurate.

Signed: Date: 24/01/2017

Catherine Paquet conceived and designed the study, made a significant contribution to the acquisition and preparation of data used in the study, provided input to the data analysis, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

Signed: Date: 25/01/2017

Natasha J. Howard made a significant contribution to the acquisition and preparation of data used in the study, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Neil T. Coffee made a significant contribution to the acquisition and preparation of data used in the study, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Anne W. Taylor made a significant contribution to the acquisition and preparation of data used in the study, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Theo Niyonsenga provided input to the data analysis, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Mark Daniel conceived and designed the study, made a significant contribution to the acquisition and preparation of data used in the study, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Authorship contributions:

Suzanne J. Carroll conceived and designed the study, analysed the data, contributed to the interpretation of the results, and wrote and approved the manuscript. I certify that the statement of contribution is accurate.

Signed: Date: 3/February/2017

Catherine Paquet conceived and designed the study, provided input to the analyses, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Natasha J. Howard contributed to interpretation of results, revised the manuscript critically for important intellectual content and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Neil T. Coffee contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Robert J. Adams revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Anne W. Taylor revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Theo Niyonsenga provided input to the data analysis, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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Mark Daniel conceived and designed the study, contributed to interpretation of results, revised the manuscript critically for important intellectual content, and approved the final manuscript. I certify that the statement of contribution is accurate and permission is given for Suzanne Carroll to include this manuscript in her thesis for examination towards the Doctor of Philosophy.

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