

Spatial distribution of the risk for metabolic complications: an application in south-east Brazil, 2006–2007

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Abstract

Objective: To identify spatial variation in the risk for metabolic complications (RMC) by means of a semi-parametric approach for multinomial data.

Design: Cross-sectional study.

Setting: We visited 730 households selected in the first of a two-stage sample in South health district in Campinas, São Paulo, Brazil, 2006–2007.

Subjects: We interviewed 651 individuals and obtained their respective anthropometric measures and geographical coordinates of their house location. They were classified according to a combination of BMI and abdominal circumference as having no risk, increased, high or very high RMC.

Results: Gender, age and schooling were associated with RMC. Crude spatial risk for the three levels of RMC in relation to the absence of risk suggested different patterns in each level. Adjusted spatial risk for the RMC showed smaller significant areas, but the pattern remained similar to crude risk.

Conclusions: Spatial point analysis with a multinomial approach improves the understanding of differences in RMC found, as we could identify specific areas in which to intervene. The public health significance of these findings may lie in the additional evidence provided that spatial location and its features can influence patterns of RMC.

Keywords
Obesity
Epidemiology
Spatial risk
Metabolic complications

The recent increase in the prevalence of obesity is widely recognized as constituting a major threat to health in most countries as obesity has reached epidemic proportions globally, with 1.5 billion adults overweight in 2008 according to the WHO⁽¹⁾. Based on data from the National Health and Nutrition Examination Survey 2007–2008, the estimated prevalence of obesity (BMI \geq 30 kg/m²) for adults aged 20 years or older in the USA was 33.8%⁽²⁾, while for adult men (women) in the UK, obesity prevalence was 22.2% (23.0%) in 2003⁽³⁾. Kelly *et al.* reported a prevalence of 1.8% (4.4%) for men (women) in India and 16.0% (20.0%) for men (women) in Latin America and the Caribbean in 2005⁽⁴⁾. In Brazil in 2002–2003, the prevalence was 8.9% for men and 13.1% for women⁽⁵⁾. Although developed countries present higher levels of obesity, developing countries also show increasing overweight prevalence, mainly as a consequence of the nutrition transition they are experiencing⁽⁶⁾. Brazil has continental dimensions and inequality is present all over the country. Trends show a shift in the prevalence from the higher to the lower socio-economic level in Brazil⁽⁷⁾

and this change has contributed to the coexistence of an overweight person with an underweight person in the same household, called the ‘dual burden’ of disease^(8–10).

The number of studies on obesity has increased significantly in the last decade, mainly due to the high risk that this condition leads to patients with other chronic diseases, such as diabetes and hypertension^(11–13). Although several individual-level risk factors for obesity have been identified, population rates of obesity are determined by a complex interplay of biological, social, environmental, behavioural and cultural factors, which collectively have created over decades an adverse environment for maintaining a healthy weight. A comprehensive understanding of how these factors interact is currently lacking^(14,15). Age, sex, socio-economic status, sedentary lifestyle and co-morbidities such as diabetes and hypertension have traditionally been studied as risk factors for overweight or obesity. However, investigations on the risk of obesity and its association with factors other than those already widely studied have been proposed. The condition of obesity is not a communicable disease,

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but studies on its spatial distribution are being developed by considering the food supply and environmental conditions in a particular area^(11,16–20). The inclusion of GIS (geographic information systems) methods for assessing the food environment is already in use⁽²¹⁾ and the study of factors influencing the difference in obesity prevalence by region raises hypotheses about the influence of the environment in this epidemic^(20,22,23).

Although BMI is the most widely used anthropometric index and cut-off points to define excess of weight are already established, other indices like waist circumference and waist-to-hip ratio also have been found to be associated with all-cause mortality, diabetes mellitus, cardiovascular morbidity and mortality in prospective studies⁽²⁴⁾. However, waist circumference alone is unsatisfactory in detecting people in need of weight management on the basis of either a high BMI and/or a high waist-to-hip ratio⁽²⁵⁾. A combination of indices indicating excess of weight and abdominal adiposity is then recommended for risk assessment for type 2 diabetes, hypertension and CVD^(26–32).

Another aspect to be considered when studying metabolic complications is the polytomous characteristic of its risk, classified as no risk, increased, high or very high risk, according to a combination of BMI and abdominal circumference. The proper way to analyse an outcome with more than two categories is by use of a multinomial model that allows to one research, in a single analysis, the association for each class observed⁽³³⁾. Although the estimates of relative risk for variables with more than two categories require a multinomial model, transforming data in several dichotomous variables is still commonly seen^(34–37). Besides, including spatial analysis in a multinomial model can provide valuable information about the risk of a specific condition. Therefore we aimed to identify spatial variation in the risk for metabolic complications by means of a semi-parametric approach for multinomial data, in order to reveal locations for public health interventions to prevent an epidemic condition.

Methods

We conducted a population-based cross-sectional study between November 2006 and December 2007 in one of five health districts of Campinas (São Paulo, Brazil) of area 128 km² and estimated population 277 000 inhabitants. The sample was selected in two stages, households and individuals, according to records of the Health Unit of the district, and comprises the South district of the city. The study sample was composed of 651 (89%) local residents of 730 visited households.

After informed consent from the selected individuals we applied a standardized questionnaire and anthropometric measures were obtained following the methods proposed by the *Anthropometric Standardization Reference Manual*⁽³⁸⁾.

Table 1 Number of men and women stratified for levels of risk for type 2 diabetes, hypertension and CVD according to the combined recommendations of BMI and abdominal circumference cut-off points⁽³¹⁾, Campinas, São Paulo, Brazil, 2006–2007

BMI (kg/m ²)	Abdominal circumference (cm)	
	≤102; ≤88 (men; women)	>102; >88 (men; women)
<18.5	No risk (3; 7)	No risk (0; 0)
18.5–24.9	No risk (131; 147)	Increased risk (0; 10)
25.0–29.9	Increased risk (94; 57)	High risk (11; 51)
≥30.0	High risk (23; 8)	Very high risk (26; 83)

Weight was measured with a portable electronic balance with a capacity up to 150 kg, height was measured using a portable stadiometer and we used an inelastic tape with a capacity of 150 cm with 0.1 cm markings to measure body circumferences. Geographical coordinates of addresses of the participants were taken using a portable GPS (Global Positioning System), datum SAD-69 and projection UTM 23S, with an average accuracy of 7 m.

We used an association of abdominal circumference and BMI as a combined form of risk assessment for type 2 diabetes, hypertension and CVD. This combined risk, called 'risk for metabolic complications' (RMC) herein, was defined according to the National Institutes of Health^(26,29–31) as shown in Table 1.

Statistical analysis

We implemented a routine based on the spatial risk functions for epidemiological studies proposed by Bithell⁽³⁹⁾ and the estimated spatial risk through Generalized Additive Models (GAM) proposed by Kelsall and Diggle⁽⁴⁰⁾. Both studies worked from a dichotomous perspective, classifying individuals into two categories (ill and not ill). Our routine considers the multinomial response to estimate the spatial risk through a GAM, a model that enables the inclusion of non-spatial covariates that may be related to the outcome being studied, called a 'semi-parametric model'. In the GAM, the non-parametric function was fitted by a bi-dimensional Nadaraya–Watson kernel.

The significance of spatial effects was obtained by a Monte Carlo method. Based on the probabilities estimated under a logistic model without the spatial component, a new response variable was built and, with it, the GAM was fitted to obtain the spatial risk estimated in a grid of points representing the study area. This was repeated 400 times and the results at each point were normally distributed. From this, the estimates based on the observed data were compared with these distributions and classified as significant if they were outside the region of tolerance ($P < 0.025$ or $P > 0.975$).

For the parametric part of the model, the odds ratio was obtained through a polytomous logistic model⁽³³⁾, considered the most appropriate analysis as we had a multinomial response with a clearly defined reference

category (no risk). Analyses were performed using the software R version 2.10 for Linux (R Project for Statistical Computing, Vienna, Austria).

Results

The sample was composed mostly of women (55.7%), mean age was 41.6 (sd 12.5) years and more than half of the sample (52.5%) had studied for >8 years. Obesity (BMI ≥ 30.0 kg/m²) was present in more than 20% of the total sample (17.0% of men and 25.1% of women). Overweight (BMI = 25.0–29.9 kg/m²) was present in about a third of the total sample (29.8% of women and 36.5% of men). Abdominal circumference values above the upper limit (102 cm for men and 88 cm for women) were found in 12.8% of men and 39.7% of women (Table 2).

A third of men presented an increased RMC (33.0%), while almost a quarter of women (22.9%) had very high RMC. Older people presented very high RMC (21.4% of those aged >55 years), although increased RMC was seen for 25% of individuals in all age categories. RMC was more frequent among those with lower levels of education (63.9%

for 0–4 years of study) than among the higher educated (51.2% for >8 years of study), as shown in Table 3.

In the multinomial semi-parametric model analysis with variables age, schooling and the geographical coordinates, age was significant for all levels of RMC. For those aged >30 years the odds of having increased RMC were fourfold greater or more compared with those aged <30 years. These effects seemed to decline as the outcome became more severe, with the odds decreasing to 1.7 for very high RMC in both age categories (30–55 years and >55 years).

More years of study were protective for the high RMC category only (Table 4). Note that gender is not included in the model because it is used in the definition of the levels of the RMC.

The house locations for the individuals of the sample are plotted according to each level of the RMC in Fig. 1. The sample covers the entire populated area.

Crude spatial risk for the three levels of the RMC (increased, high and very high) in relation to absence of risk suggested different patterns in each level (Fig. 2). We detected a significant area in the north-east of the region for both increased and high categories of the

Table 2 Distribution of BMI and abdominal circumference among adult men and women, Campinas, São Paulo, 2006–2007

	Men (n 288)		Women (n 363)		Total (n 651)	
	%	n	%	n	%	n
BMI (kg/m ²)						
<25.0	46.5	134	45.2	164	45.8	298
25.0–29.9	36.5	105	29.8	108	32.7	213
≥ 30.0	17.0	49	25.1	91	21.5	140
Abdominal circumference (cm)						
<102 (Male)	87.2	251	–	–	72.2	470
<88 (Female)	–	–	60.3	219	–	–
≥ 102 (Male)	12.8	37	–	–	27.8	181
≥ 88 (Female)	–	–	39.7	144	–	–

Table 3 Prevalence of risk for metabolic complications (RMC) according to characteristics of the sample, Campinas, São Paulo, 2006–2007

	RMC				P value*
	No risk (n 288)	Increased (n 162)	High (n 92)	Very high (n 109)	
	%	%	%	%	
Gender					
Men	46.5	33.0	11.5	9.0	
Women	42.4	18.5	16.3	22.9	<0.001
Age (years)					
<30	60.8	24.1	7.6	7.6	
30–55	38.8	25.5	16.5	19.2	
>55	39.3	24.1	15.2	21.4	<0.001
Schooling (years of study)					
0–4	36.1	24.7	19.6	19.6	
5–8	42.4	26.5	15.2	15.9	
>8	48.8	24.3	11.1	15.8	0.086
Total	44.2	24.9	14.1	16.7	

*P values for Pearson's χ^2 association test of significance.

Table 4 Semi-parametric model for risk for metabolic complications (RMC), Campinas, São Paulo, 2006–2007

	RMC					
	Increased		High		Very high	
	OR	95% CI	OR	95% CI	OR	95% CI
Age (years)						
<30	Ref.		Ref.		Ref.	
30–55	3.89	2.23, 6.80	3.08	1.78, 5.32	1.66	1.21, 2.28
>55	4.74	2.52, 8.92	2.64	1.36, 5.13	1.66	1.08, 2.54
Schooling (years of study)						
0–4	Ref.		Ref.		Ref.	
5–8	0.85	0.54, 1.32	0.83	0.53, 1.30	1.16	0.81, 1.66
>8	0.76	0.52, 1.11	0.56	0.37, 0.84	0.87	0.63, 1.21

Ref., reference category.
All RMC categories compared with no risk.

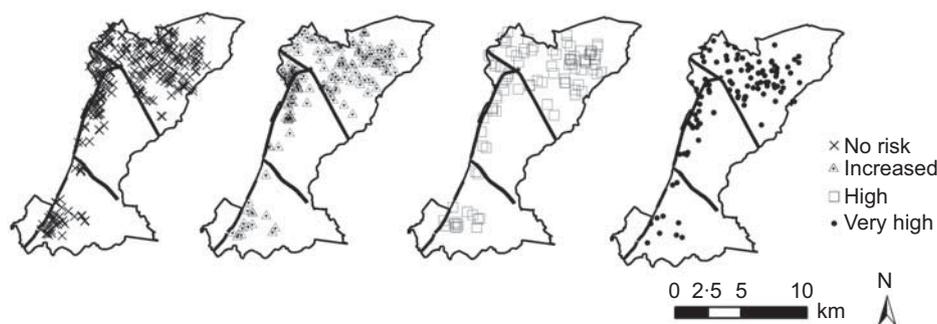


Fig. 1 Distribution of a population-based sample according to risk for metabolic complications, Campinas, São Paulo, Brazil, 2006–2007

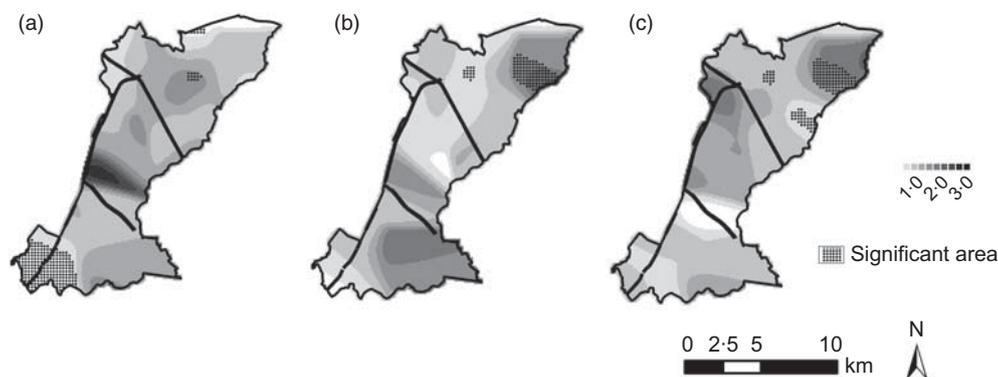


Fig. 2 Crude spatial analysis of the risk for metabolic complications, Campinas, São Paulo, Brazil, 2006–2007: (a) increased; (b) high; (c) very high

RMC (Figs 2(a) and (b)). We also found a small area with lower odds for increased RMC in the east of the region (Fig. 2(a)) and for high RMC in the central north (Fig. 2(b)). For very high RMC we found a protective area in the south-west of the region (Fig. 2(c)).

As shown in Table 3, gender, age and schooling might influence the distribution of RMC over the region. So, an adjusted spatial risk for the RMC in relation to the absence of risk was calculated (Fig. 3). As expected, the surface of the adjusted risk was more flat compared with Fig. 2 and

the significant areas were smaller, although the patterns of the risk remained similar.

Discussion

The analytical method proposed here considers all levels of the response variable in a single model and the inclusion of a non-parametric component, the geographical coordinates, as an alternative to study the influence of



Fig. 3 Adjusted spatial analysis of the risk for metabolic complications, Campinas, São Paulo, Brazil, 2006–2007: (a) increased; (b) high; (c) very high

environment on the risk for type 2 diabetes, hypertension and CVD. The results presented indicate that the association of house location and RMC calls for more consideration. These population-based data for an urban region of 277 000 inhabitants and area of 128 km² showed few significant associations considering both crude and adjusted spatial risk. Age >30 years was associated with RMC in all three of its categories, while more years of study was protective for RMC only in the high risk category. Higher prevalence of overweight and obesity with increasing age and lower education levels has also been detected in Brazil by Monteiro *et al.*⁽⁷⁾.

Urban characteristics in the region point to the similarity in the prevalence of obesity and consequently less variation of the RMC. The small area detected as statistically significant for increased RMC has better socio-economic indicators than the whole region, and the protective area corresponds to lower indices as income, schooling of household heads and urban infrastructure (water supply, sewage and garbage collection). For very high RMC, the protective area corresponds to a very poor county⁽⁴¹⁾.

Although we had a lack of environmental information such as food supply and access to parks, we could identify an association between RMC and areas with certain sociodemographic characteristics which corroborates the consensus reported by the US Institute of Medicine⁽¹⁴⁾ stating that the environmental factors play a role and that environmental solutions are required to address the epidemic of obesity⁽¹⁵⁾. In this direction, it is noted also that the nutritional environment has been investigated in order to deepen the understanding of the widespread prevalence of obesity, including the study of local availability, access to health food and environmental conditions^(42–45). These data support the importance of focusing on places in addition to the individuals who live in those places. The public health significance of these findings may lie in the additional evidence provided that spatial location and its features can influence patterns of risk for type 2 diabetes, hypertension and CVD.

Multinomial logistic regression is already widely used in epidemiological studies and can identify different patterns in the levels or categories of the response variable^(46,47). The selection of GAM with polytomous logistic regression and bi-dimensional kernel enriches the present analysis and provides another way to analyse data on the epidemic of obesity, a challenge for public health today. Because this epidemic is the result of a system that contains a diverse set of factors at many levels of scale, with different individual motivations and priorities, the search for new forms of analysis that can address all of the factors at their different levels helps in its understanding, combat and prevention⁽⁴⁸⁾.

One limitation of the present study, and indeed most of studies on this topic, is that our data are observational and cross-sectional. Because of this, causal association of current house location with RMC cannot be attributed to the location and the variables included in the statistical model. Additionally, we did not include information about diet or lifestyle variables in the analysis as the main objective was to apply the multinomial method and to locate the individuals at risk. A further limitation is that although the sample size was adequate for the statistical analysis and we covered the entire inhabited area, we found similarity in the region. The features of the region, being predominantly urban with similar economic and social development throughout, hampered the identification of an environmental component that could be influencing the differences in risks within the area. Strengths of the present study include greater precision in the measurement of individual location to estimate the spatial risk over the region and the use of the semi-parametric GAM combining this location with other covariates of interest.

While there are many ways to define community, geographic location is one important way to understand the context in which people live⁽²³⁾. We identified small areas with different levels of risk, allowing health interventions in the community and providing information for community-based health research. Future studies

including prospective follow-up, evaluation of community interventions and additional information that has been studied elsewhere^(15,16,20,23,49–52), such as food supply, access to parks, diet and physical activity, will help understand the growth of the obesity epidemic in developed countries. In order to establish associations between the obesogenic environment, eating patterns and health, we need good and reliable tools and indicators to characterize these environments⁽⁵²⁾. In previous centuries, major advances in the control of infectious diseases, like cholera and tuberculosis, came from environmental changes involving public sanitation. The modern epidemics of obesity, type 2 diabetes, hypertension and CVD may require a return to this basic strategy. Studies suggest that community redevelopment and housing policy, in addition to other benefits, may contribute to improvements in population health⁽¹⁵⁾.

Conclusions

Spatial point analysis with a multinomial approach to properly treat variables with more than two categories improves the quality of data information. Risk and protective areas for metabolic complications were identified despite the distribution of the population according to gender, age and schooling. Thorough investigation is needed to identify the environmental factors that are responsible for the higher and lower risk in the studied area, but our results lead to a rapid intervention as was done for transmissible diseases in the past.

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References

1. World Health Organization (2011) *Obesity and Overweight*. Geneva: WHO; available at <http://www.who.int/mediacentre/factsheets/fs311/en/index.html>
2. Flegal KM, Carroll MD, Ogden CL *et al.* (2010) Prevalence and trends in obesity among US adults, 1999–2008. *JAMA* **303**, 235–241.
3. Berghofer A, Pischon T, Reinhold T *et al.* (2008) Obesity prevalence from a European perspective: a systematic review. *BMC Public Health* **8**, 200.
4. Kelly T, Yang W, Chen CS *et al.* (2008) Global burden of obesity in 2005 and projections to 2030. *Int J Obes (Lond)* **32**, 1431–1437.

5. Instituto Brasileiro de Geografia e Estatística (2004) *Pesquisa de Orçamentos Familiares 2002–2003. Análise da disponibilidade domiciliar de alimentos e do estado nutricional no Brasil*. Rio de Janeiro: IBGE.
6. Popkin BM (1998) The nutrition transition and its health implications in lower-income countries. *Public Health Nutr* **1**, 5–21.
7. Monteiro CA, Conde WL & Popkin BM (2004) The burden of disease from undernutrition and overnutrition in countries undergoing rapid nutrition transition: a view from Brazil. *Am J Public Health* **94**, 433–434.
8. Doak CM, Adair LS, Bentley M *et al.* (2005) The dual burden household and the nutrition transition paradox. *Int J Obes (Lond)* **29**, 129–136.
9. Caballero B (2005) A nutrition paradox – underweight and obesity in developing countries. *N Engl J Med* **352**, 1514–1516.
10. Caballero B (2007) The global epidemic of obesity: an overview. *Epidemiol Rev* **29**, 1–5.
11. Mokdad AH, Bowman BA, Ford ES *et al.* (2001) The continuing epidemics of obesity and diabetes in the United States. *JAMA* **286**, 1195–1200.
12. Resta O, Foschino-Barbaro MP, Legari G *et al.* (2001) Sleep-related breathing disorders, loud snoring and excessive daytime sleepiness in obese subjects. *Int J Obes Relat Metab Disord* **25**, 669–675.
13. Peeters A, Barendregt JJ, Willekens F *et al.* (2003) Obesity in adulthood and its consequences for life expectancy: a life-table analysis. *Ann Intern Med* **138**, 24–32.
14. Koplan JP, Liverman CT & Kraak VA (2005) *Preventing Childhood Obesity: Health in the Balance*. Washington, DC: Institute of Medicine of the National Academies.
15. Glass TA, Rasmussen MD & Schwartz BS (2006) Neighborhoods and obesity in older adults: the Baltimore Memory Study. *Am J Prev Med* **31**, 455–463.
16. Hill JO & Peters JC (1998) Environmental contributions to the obesity epidemic. *Science* **280**, 1371–1374.
17. Young LR & Nestle M (2002) The contribution of expanding portion sizes to the US obesity epidemic. *Am J Public Health* **92**, 246–249.
18. Holsten JE (2009) Obesity and the community food environment: a systematic review. *Public Health Nutr* **12**, 397–405.
19. Zangirolani LTO (2009) *Topologia do excesso de peso no Distrito Sul de Campinas, São Paulo*. Campinas: State University of Campinas.
20. Drewnowski A, Rehm CD & Solet D (2007) Disparities in obesity rates: analysis by ZIP code area. *Soc Sci Med* **65**, 2458–2463.
21. Charreire H, Casey R, Salze P *et al.* (2010) Measuring the food environment using geographical information systems: a methodological review. *Public Health Nutr* **13**, 1773–1785.
22. Ouedraogo HZ, Fournet F, Martin-Prevel Y *et al.* (2008) Socio-spatial disparities of obesity among adults in the urban setting of Ouagadougou, Burkina Faso. *Public Health Nutr* **11**, 1280–1287.
23. Schlundt DG, Hargreaves MK & McClellan L (2006) Geographic clustering of obesity, diabetes, and hypertension in Nashville, Tennessee. *J Ambul Care Manage* **29**, 125–132.
24. Can AS, Bersot TP & Gonen M (2009) Anthropometric indices and their relationship with cardiometabolic risk factors in a sample of Turkish adults. *Public Health Nutr* **12**, 538–546.
25. Molarius A, Seidell JC, Sans S *et al.* (1999) Varying sensitivity of waist action levels to identify subjects with overweight or obesity in 19 populations of the WHO MONICA Project. *J Clin Epidemiol* **52**, 1213–1224.
26. Associação Brasileira para o Estudo da Obesidade e da Síndrome Metabólica (2009) *Diretrizes brasileiras de obesidade 2009/2010/ABESO – Associação Brasileira para o Estudo da Obesidade e da Síndrome Metabólica*, 3rd ed. Itapevi, SP: AC Farmacêutica.

27. World Health Organization (1995) *Physical Status: The Use and Interpretation of Anthropometry*. Geneva: WHO.
28. Zhu S, Heshka S, Wang Z *et al.* (2004) Combination of BMI and waist circumference for identifying cardiovascular risk factors in whites. *Obes Res* **12**, 633–645.
29. Rexrode KM, Carey VJ, Hennekens CH *et al.* (1998) Abdominal adiposity and coronary heart disease in women. *JAMA* **280**, 1843–1848.
30. International Diabetes Federation (2006) *The IDF Consensus Worldwide Definition of the Metabolic Syndrome*, p. 24. Brussels: IDF Communications.
31. North American Association for the Study of Obesity & National Heart, Lung, and Blood Institute (2000) *The Practical Guide: Identification, Evaluation, and Treatment for Overweight and Obesity in Adults*. Bethesda, MD: US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Heart, Lung, and Blood Institute.
32. World Health Organization (2011) *Waist Circumference and Waist–Hip Ratio: Report of a WHO Expert Consultation, Geneva, 8–11 December 2008*. Geneva: WHO.
33. Ananth CV & Kleinbaum DG (1997) Regression models for ordinal responses: a review of methods and applications. *Int J Epidemiol* **26**, 1323–1333.
34. Hasselmann MH & Reichenheim ME (2006) Parental violence and the occurrence of severe and acute malnutrition in childhood. *Paediatr Perinat Epidemiol* **20**, 299–311.
35. Hindorff LA, Lemaitre RN, Smith NL *et al.* (2008) Common genetic variation in six lipid-related and statin-related genes, statin use and risk of incident nonfatal myocardial infarction and stroke. *Pharmacogenet Genomics* **18**, 677–682.
36. Mavaddat N, Dunning AM, Ponder BA *et al.* (2009) Common genetic variation in candidate genes and susceptibility to subtypes of breast cancer. *Cancer Epidemiol Biomarkers Prev* **18**, 255–259.
37. Zammit W, Mtraoui N, Kallel C *et al.* (2006) A case-control study on the association of idiopathic recurrent pregnancy loss with autoantibodies against β_2 -glycoprotein I and annexin V. *Reproduction* **131**, 817–822.
38. Lohman T, Roche A & Martorell R (1991) *Anthropometric Standardization Reference Manual*. Champaign, IL: Human Kinetics Books.
39. Bithell JF (1990) An application of density estimation to geographical epidemiology. *Stat Med* **9**, 691–701.
40. Kelsall JE & Diggle PJ (1998) Spatial variation in risk of disease: a nonparametric binary regression approach. *J R Stat Soc (Ser C) Appl Stat* **47**, 559–573.
41. Sistema de Informação TabNet (2000) Campinas: Coordenadoria de Informação e Informática da secretaria de Saúde de Campinas. <http://tabnet.saude.campinas.sp.gov.br/> (accessed November 2010).
42. Glanz K, Sallis JF, Saelens BE *et al.* (2005) Healthy nutrition environments: concepts and measures. *Am J Health Promot* **19**, 330–333.
43. Michimi A & Wimberly MC (2010) Spatial patterns of obesity and associated risk factors in the conterminous US. *Am J Prev Med* **39**, e1–e12.
44. Poulou T & Elliott SJ (2009) An exploratory spatial analysis of overweight and obesity in Canada. *Prev Med* **48**, 362–367.
45. Procter KL & Smith DM (2008) Size matters: the role of scale in geographies of health. *Area* **40**, 303–305.
46. Kilicarslan A, Isildak M, Guven GS *et al.* (2006) Demographic, socioeconomic and educational aspects of obesity in an adult population. *J Natl Med Assoc* **98**, 1313–1317.
47. Ziraba AK, Fotso JC & Ochako R (2009) Overweight and obesity in urban Africa: a problem of the rich or the poor? *BMC Public Health* **9**, 465.
48. Hammond RA (2009) Complex systems modeling for obesity research. *Prev Chronic Dis* **6**, A97.
49. Sharkey JR, Horel S, Han D *et al.* (2009) Association between neighborhood need and spatial access to food stores and fast food restaurants in neighborhoods of colonias. *Int J Health Geogr* **8**, 9.
50. Song HJ, Gittelsohn J, Kim M *et al.* (2009) A corner store intervention in a low-income urban community is associated with increased availability and sales of some healthy foods. *Public Health Nutr* **12**, 2060–2067.
51. Li F, Harmer P, Cardinal BJ *et al.* (2009) Obesity and the built environment: does the density of neighborhood fast-food outlets matter? *Am J Health Promot* **23**, 203–209.
52. Elinder LS & Jansson M (2009) Obesogenic environments – aspects on measurement and indicators. *Public Health Nutr* **12**, 307–315.