Journal of Public Health | Vol. 38, No. 2, pp. e39–e46 | doi:10.1093/pubmed/fdv107 | Advance Access Publication 19 August 2015

Socioeconomic position in young adulthood is associated with BMI in Australian families

Katrina J. Scurrah[1,2], Anne M. Kavanagh[3], Rebecca J. Bentley[3], Lukar E. Thornton[4], Stephen B. Harrap[1]

[1]Department of Physiology, The University of Melbourne, Melbourne, VIC, Australia
[2]Australian Centre of Excellence in Twin Research, Centre for Epidemiology and Biostatistics, Melbourne School of Population and Global Health, The University of Melbourne, Melbourne, VIC, Australia
[3]Gender and Women's Health Unit, Centre for Health Equity, Melbourne School of Population and Global Health, The University of Melbourne, Melbourne, VIC, Australia
[4]Centre for Physical Activity and Nutrition Research, School of Exercise and Nutrition Sciences, Deakin University, Melbourne, VIC, Australia
Address correspondence to Katrina Scurrah, E-mail: kscurrah@unimelb.edu.au

ABSTRACT

Background Low socioeconomic position (SEP) is associated with increased cardiovascular (CV) disease risk, but the relative importance of SEP in childhood and adulthood, and of changes in SEP between these two life stages, remains unclear. Studies of families may help clarify these issues. We aimed to assess whether SEP in young adulthood, or change in SEP from childhood to young adulthood, was associated with five continuously measured CV risk factors.

Methods We used data from 286 adult Australian families from the Victorian Family Heart Study (VFHS), in which some offspring have left home ($n = 364$) and some remained at home ($n = 199$). SEP (defined as the Index of Relative Socioeconomic Disadvantage) was matched to addresses. We fitted variance components models to test whether young adult SEP and/or change in SEP was associated with systolic blood pressure, diastolic blood pressure, body mass index (BMI), total cholesterol or high-density lipoprotein cholesterol, after adjustment for parental SEP and within-family correlation.

Results An increase in SEP of 100 SEIFA units from childhood to adulthood was associated with a lower BMI ($\beta = -0.49$ kg/m^2, $P < 0.01$) only.

Conclusions These results suggest that a change in SEP in young adulthood is an important predictor of BMI, independent of childhood SEP.

Keywords blood pressure, cardiovascular risk, cardiovascular risk factors, family study, within-family correlation, young adults

Introduction

Cardiovascular (CV) disease is a major global cause of death and disability. Considered multifactorial in origin, CV disease represents the singular and combined effects of genes and environment. Low socioeconomic position (SEP) in adulthood has consistently been associated with deaths from CV disease and CV risk factors such as body mass index (BMI), blood pressure and cholesterol.[1–6]

Barker and others[7–10] have pointed to the importance of environmental exposures in early life and their impact on adult CV disease. Early life SEP correlates with adult CV disease and risk factors. However, there is uncertainty as to the relative importance of SEP in adulthood for CV risk independent of early life SEP, as the two are correlated[11] and few studies have the appropriate data to account for early life SEP. If adult SEP has little proportional influence on CV risk, over and above early life SEP, then efforts should be directed towards understanding and, indeed, intervening in early life exposures.

Appropriate study design is key to adequately accounting for early life SEP and accounting for factors that cannot be

Katrina J. Scurrah, Senior Research Fellow (Statistician)
Anne M. Kavanagh, Professor of Women's Health
Rebecca J. Bentley, Senior Lecturer
Lukar E. Thornton, Senior Research Fellow
Stephen B. Harrap, Professor of Physiology

measured but are likely to be confounding observed relationships between adult SEP and CV disease. Families provide a useful setting in which to address this issue because family members will have shared childhood exposure to SEP and other factors. Family members who have changed their SEP circumstances in adult life provide a useful source of comparison with members who remain in the same socioeconomic setting in which they grew up. Most previous studies have investigated relationships between SEP and CV risk factors in older adults, but assessment of the relationships in young adults would allow investigation of whether or not changes occur quickly after leaving home. The Victorian Family Heart Study (VFHS) comprises adult, two-generation families in which some young adult offspring moved away from the home environment. The aim of this study was to determine whether SEP in young adults is associated with CV risk factors, after adjustment for parental SEP and unmeasured shared familial genetic and environmental effects.

Methods

Study population

The details of the recruitment of subjects for the VFHS have been published previously.[12] In brief, a volunteer sample of 767 Caucasian adult families enriched with families containing twins (65 monozygotic pairs, 84 dizygotic pairs) was recruited from a variety of community-based sources. A family history of heart disease was not a prerequisite for recruitment. Families comprised both parents aged between 40 and 70 years and at least one natural offspring aged between 18 and 30 years.

The Ethics Review Committee of the Alfred Hospital, Melbourne approved the study and informed consent was obtained from all participants. Participants attended research clinics where trained research nurses obtained relevant information regarding drug treatment and smoking, measured CV and other outcomes such as height and weight, and took blood samples as detailed previously.[12]

After resting for 10 min, three measurements of both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken in the supine position, the last two of which were recorded. Subjects then stood for 2 min and three further measurements of SBP and DBP were taken, the last two of which were recorded. For subjects receiving antihypertensive treatments, we adjusted the recorded pressures by adding 10 and 5 mmHg to SBP and DBP, respectively, as justified previously.[13,14] In this study, lying and standing measurements were averaged.

Following phenotypic measurements, venous blood was collected for biochemical analysis. After insertion of a butterfly needle, the tourniquet was released before collection of 7 ml of blood into lithium heparin anticoagulant. Total cholesterol and high-density lipoprotein (HDL) cholesterol were measured by automated biochemical analysis systems.

Offspring whose recorded addresses were the same as their parents' were classified as 'dependent' individuals; all other offspring were classified as 'independent'. This is because we assume the group not living at the same address as their parents are responsible for their own diets and lifestyle.

Socioeconomic indices

The Australian Bureau of Statistics (ABS) uses principal components analysis to derive five Socio-Economic Indexes for Areas (SEIFA, or SEIFA indexes) based on census data.[15] We used the most commonly used Index of Relative Socio-Economic Disadvantage (IRSD), which is derived from attributes such as low income, low educational attainment and high unemployment. Lower IRSD scores reflect more socioeconomically disadvantaged areas.

The IRSD score assigned to respondents was based on their census collector district (CCD). CCD boundaries from 1996 were used and at this time a CCD was the smallest geographic area used for census data output. Each CCD included 225 dwellings on average in urban areas[16] and there were 7889 Victorian CCDs in total.[15] CCD level SEIFA measures are commonly used as a proxy for individual SEP in Australian studies and have consistently been found to demonstrate strong socioeconomic gradients, provide a good reflection of individuals' SEP[17] and be highly correlated with individual behavioural factors such as physical activity levels.[18]

Geocoding methods

Geocoding of all 2999 participants in the VFHS was undertaken using ArcGIS (v 9.1).[19] Initial geocoding resulted in a 90% match. The remaining 270 unmatched cases were mainly due to spelling errors or addresses on suburb borders. In the cases ($n = 42$) where it could be determined where the respondent lived but no address point existed, they were geocoded to the next point provided there was no ambiguity as to which CCD the address point should be within. This final interactive match resulted in only 70 (2%) study participants being unmatched. Many common geocoding errors were avoided[20] using this meticulous process.

Addresses were matched to their corresponding CCD, using a spatial join of the geocoded address points and the CCD polygons. SEIFA IRSD scores for the respondents' CCD were then joined to the geocoded address table, which was then exported for analysis.

For independent offspring, two SEIFA IRSD variables were available: IRSD based on the CCD of their current address (referred to as current or adult SEP), and the IRSD based on the CCD of their father's current address (referred to

as parental IRSD or parental SEP). For dependent offspring, own IRSD was identical to parent IRSD.

Study sample

Both CV risk factor and IRSD data were available for 1135 individuals (including parents) in 286 families where both parents lived at the same address but at least one adult offspring had a different address from that of his/her parents. There were 220 families with one independent offspring, 55 families with two independent offspring and 11 families with three or four independent offspring. One hundred 'families' consisted of only one independent young adult. In total, these families comprised 563 offspring (364 independent and 199 dependent). Included in the dataset were 20 pairs of MZ twins (2 dependent pairs, 10 independent pairs and 8 discordant pairs) and 41 pairs of DZ twins (14 dependent pairs, 15 independent pairs and 12 discordant pairs). Of the 385 total pairs, 211 were same-sex pairs and 174 were opposite-sex pairs.

Statistical methods

SEP within families: general approach

To assess the association of SEP, and change in SEP, with CV risk factors within families, variance components models were fitted using maximum likelihood estimation in the software package Solar[21] to the five outcomes: SBP, DBP, BMI, total cholesterol and HDL cholesterol.

Fixed covariates included age (centred), sex, and age–sex interaction (included in all models). The basic fixed effects model for each individual j within family i was as follows:

$$\hat{y}_{ij} = \beta_0 + \beta_{\text{age}} \times \text{age}_{ij} + \beta_{\text{sex}} \times \text{Male}_{ij} + \beta_{\text{age*sex}} \times \text{Male}_{ij} \times \text{age}_{ij}$$

where $\hat{y}_{ij}$ is the expected value for each CV risk factor, betas are regression coefficients and the variable Male_{ij} takes the value 1 if individual j within family i is male and 0 otherwise.

Within each family (sibship) i, a multivariate normal distribution was assumed for the vector of outcomes y_i conditional on covariates; $\underline{y}_i \sim N(\beta^T X_i, V_i)$, where β^T is the vector of regression coefficients, X_i is the covariate matrix and V_i is the covariance matrix, with elements $V_{i,jk}$. For some models, a multivariate t distribution was assumed, and in this case $\underline{y}_i \sim \text{MVT}(\beta^T X_i, V_i, \text{d})$, where d is the number of degrees of freedom of the t distribution and is estimated during model fitting.

The general form of the covariance between nuclear family relatives j and k was taken to be V_{jk} defined by the following:

$$V_{jk} = \begin{cases} \rho_{jk}\sigma^2, & j \neq k \\ \sigma^2, & j = k \end{cases}$$

where ρ_{jk} is one of ρ_{SIB}, ρ_{DZ} and ρ_{MZ}, depending on whether j and k are from a sibling, DZ or MZ twin pair. In some models, these three correlations were constrained to be equal. This general model has been used for previous analyses of data from this study.[12]

SEP change in dependent and independent offspring

The variance components models described above were used to assess association of the exposure 'current IRSD minus childhood IRSD' (referred to as 'SEPchange') with CV by including this exposure in the model for the mean. For dependent offspring, the value of SEPchange was equal to 0. The analyses also adjusted for parental IRSD by including this as a covariate in the model. Both SEPchange and parental IRSD were modelled as either continuous (linear effect) or categorical (three tertiles) to allow for detection of non-linear effects. Dependency status (dependent = 0, independent = 1) was also included in the model for the mean in this analysis to allow for differences in means between dependent and independent individuals.

By including both independent and dependent siblings from the same families, we were able to model shared but unmeasured early life environmental effects, shared but unmeasured genetic effects, one shared measured early life effect (parental IRSD) and one measured effect from young adulthood (current IRSD). The models can also account for several different relationship types (MZ twin pairs, DZ twin pairs and sibling pairs).

Results

In the complete VFHS cohort, the unadjusted average IRSD was 1049.0 (SD = 84.0). ISRD values are centred around 1000, indicating that the VFHS was of slightly higher SEP than that of the general community. We have previously reported that the CV risk profile of VFHS was closely representative of the community as ascertained through the Australian National Heart Foundation Risk Factor Prevalence Survey at the same period. In the complete VFHS sample (767 2 generation families), after adjustment for age, sex, generation and the two-way and three-way interactions of these, IRSD was strongly linearly associated with BMI ($\beta = -0.58$, $P < 0.001$), total cholesterol ($\beta = -0.061$, $P = 0.009$) and DBP ($\beta = -0.59$, $P = 0.012$), weakly associated with SBP ($\beta = -0.59$, $P = 0.080$) and not associated with HDL cholesterol ($\beta = 0.088$, $P = 0.20$).

The characteristics of the parents and offspring in the main analyses were representative of the complete VFHS (see[12] for full VFHS results) and are summarized in Table 1. Among the offspring, independent offspring were more often female and older than dependent offspring (Table 1). The mean difference

Table 1 Demographics of study participants and summary statistics for IRSD

Variable	*Mean (SD) for:*			
	Parents (n = 572)	*All offspring (n = 563)*	*Offspring living at home (n = 199)*	*Offspring living away from home (n = 364)*
Age	56.00 (5.67)	25.98 (3.21)	24.01 (3.08)	27.07 (2.72)
Sex (proportion of males)	0.50	0.45	0.54	0.40
Height (cm)	167.64 (8.93)	170.87 (9.45)	172.12 (9.45)	170.22 (9.24)
SBP (mmHg)	130.51 (16.83)	117.74 (11.21)	118.78 (11.40)	117.23 (11.01)
DBP (mmHg)	81.56 (9.75)	70.86 (9.22)	71.03 (9.72)	70.76 (9.06)
BMI (kg/m^2)	26.36 (4.03)	23.33 (3.41)	23.02 (3.22)	23.53 (3.51)
Total cholesterol (g/l)	5.86 (1.02)	4.79 (0.86)	4.67 (0.93)	4.87 (0.82)
HDL cholesterol (g/l)	1.42 (0.58)	1.38 (0.40)	1.38 (0.41)	1.38 (0.40)
Own IRSD	1042.51 (79.42)	1043.27 (75.92)	1054.82 (73.01)	1036.96 (76.83)
Parental IRSD		1046.55 (77.81)	1054.82 (73.01)	1041.33 (79.98)

between current SEP and parental SEP was −4.4 SEIFA units for independent offspring (Table 1), indicating that the independent young adults in the study tended to move to areas with lower SEP than their parents' current home. This change in SEP associated with leaving home was distributed such that 43% of independent young adults moved to areas with IRSD more than 25 SEIFA units lower than their parents' home, 21% stayed in areas with similar IRSD (<25 SEIFA units absolute difference) and 36% moved to areas with IRSD more than 25 SEIFA units higher than their parents' current home.

Current SEP was strongly inversely associated with BMI (Table 2) in all and in independent offspring, and the association in independent offspring remained after adjustment for parental SEP. No other linear (Table 2) or categorical (Supplementary data, Table S1) associations of SEP with CV risk factors were detected.

Table 3 shows associations between CV risk factors and change in SEP. The linear modelling revealed an inverse association between BMI and change in SEP, with a 100 unit increase in SEP from the family home and current environment associated with a 0.49 kg/m^2 decrease in BMI ($P = 0.008$, Table 3). The categorical analyses for BMI revealed a similar but weaker trend ($P = 0.063$, Table 3). Change in SEP was not associated with any of the other 4 CV risk factors, when included as either a continuous or a three-level categorical exposure (all $P > 0.25$, Table 3).

Discussion

Main findings of this study

We first estimated the association between current SEP and CV risk factors in adult offspring, focusing on those adult offspring who had left home, and then we analysed social mobility in all offspring to determine the association between changes in SEP and CV risk factors. The overall findings were consistent and revealed independent effects of SEP in early adulthood on BMI after adjustment for familial influences, particularly the parental (and assumed childhood) SEP. However, no significant independent effects of adult SEP on other CV risk factors were observed.

What is already known on this topic

SEP is associated with CV risk, but there is some inconsistency among the published twin and family studies regarding the influence of adult SEP. These studies differ in the modelling approach, measured outcomes, ages of participants, sample size, the precise measures of SEP and adjustments for relevant covariates, in particular childhood SEP. Most previous within-family studies were performed in the USA[22–25] and the Scandinavian countries,[26–30] with one study of twins from the UK,[31] one Chinese study,[32] one Indian study[33] and one Australian study.[34] All the above studies of twins and siblings have focused on SEP and CV risk in middle or older age. Previous studies have used either adult education or adult occupation as the main SEP measure, treated as a continuous (linear or non-linear), binary or ordinal exposure. Krieger *et al.* found that results differed for different indicators of SEP, with stronger results for occupation than for education.[23] Within-family effects have also been modelled in a variety of ways, including differences models, generalized estimating equations models, propensity matching and random effects models.

Some twin studies have been unable to detect significant differences in BMI for twins discordant for their adult SEP[30]

Table 2 Association of current SEP treated as continuous (linear) exposure with CV risk factors

CV risk factors	*All offspring*	*Dependent offspring*	*Independent offspring (unadjusted)*	*Independent offspring (adjusted for parental SEP)*
SBP (mmHg)	−0.23	0.65	−0.64	−0.77
95% CI	(−1.37, 0.91)	(−1.33, 2.63)	(−2.00, 0.72)	(−2.17, 0.62)
P	0.69	0.52	0.36	0.28
DBP (mmHg)	−0.33	1.25	−1.05	−0.97
95% CI	(−1.33, 0.67)	(−0.45, 2.95)	(−2.22, 0.12)	(−2.17, 0.22)
P	0.51	0.15	0.079	0.11
BMI (kg/m^2)	−0.47	0.05	−0.71	−0.70
95% CI	(−0.81, −0.13)	(−0.52, 0.62)	(−1.11, −0.31)	(−1.1, −0.29)
P	0.006	0.87	<0.001	0.001
Total cholesterol (g/l)	−0.05	−0.05	−0.05	−0.05
95% CI	(−0.14, 0.03)	(−0.21, 0.10)	(−0.15, 0.05)	(−0.16, 0.05)
P	0.23	0.48	0.32	0.32
HDL cholesterol (g/l)	−0.01	0.05	−0.04	−0.01
95% CI	(−0.28, 0.26)	(−0.40, 0.49)	(−0.35, 0.28)	(−0.33, 0.31)
P	0.93	0.84	0.81	0.95

Table 3 Association of SEP change (current minus parental SEP) and CV risk factors.

CV risk factor	*Beta-coefficient for linear association (per 100 SEIFA units)*	*Effect of SEP tertile*		*LRT* P-value
		Low- versus mid-tertile	*High- versus mid-tertile*	
SBP (mmHg)	−0.53 (−1.78, 0.71) 0.40	−0.54 (−3.28, 2.20) 0.70	−1.72 (−4.66, 1.21) 0.25	0.48
DBP (mmHg)	−0.35 (−1.43, 0.73) 0.53	0.10 (−2.27, 2.46) 0.94	−0.30 (−2.85, 2.25) 0.82	0.94
BMI (kg/m^2)	−0.49 (−0.86, −0.13) 0.008	0.64 (−0.17, 1.44) 0.12	−0.20 (−1.05, 0.65) 0.64	0.063
Total cholesterol (g/l)	−0.054 (−0.15, 0.04) 0.25	0.004 (−0.21, 0.22) 0.97	−0.07 (−0.29, 0.15) 0.54	0.73
HDL cholesterol (g/l)	0.027 (−0.27, 0.32) 0.86	0.14 (−0.48, 0.75) 0.66	0.45 (−0.22, 1.12) 0.19	0.39

Numbers in table are estimates, SEs, *P*-values. The middle tertile, [−25, 25], has been chosen as the reference since it includes all dependent offspring and is the largest category. All estimates are in comparison with this category.

or education level.[22,25] In other studies,[22,24,26,28,30,35] the effects of adult SEP were reduced or became non-significant when adjustment for shared early life effects was made (in sibpairs, twinpairs or using propensity score matching), suggesting that factors shared by siblings may explain at least some of the effects of SEP on adult CV. However, other authors[25,27,36] have concluded that such factors explain only a minor part of the association.

What this study adds

By studying young adult sibling pairs discordant for current SEP, we found that current SEP was strongly associated with BMI after adjusting for childhood SEP and unmeasured factors shared within families, but was not associated with blood pressure or cholesterol. This suggests that adult SEP is important and that the strength and/or timing of the effect varies between CV risk factors.

It is worth reflecting on the relative magnitude of the estimated effect of adult SEP on BMI. Other studies have noted that the variation in adult BMI as a result of disparity in SEP is less than the variation that can be attributed to secular trends[37] and in the case of twins, that which can be attributed to their zygosity.[30] In our analyses, we estimated an effect of 0.5 kg/m^2 of BMI for 1 SD change in current (early adulthood) SEP. In contrast, there was no effect of parental SEP on offspring BMI. The effect of current SEP was of similar magnitude (albeit in the opposite direction) to the effect of parental BMI on offspring BMI (Fig. 1A). This suggests the influence of early adulthood SEP is relatively large in the familial analysis of BMI. The same is not true for SBP, where there exists a significant influence of parental SBP but no apparent association with current SEP (Fig. 1B).

The discrepancy between various CV risk factors suggests that the influence of SEP in early adulthood varies in either the strength or timing of the effect. Blood pressure and cholesterol might be less susceptible to SEP or their effects might take longer to become apparent. For example, environmental

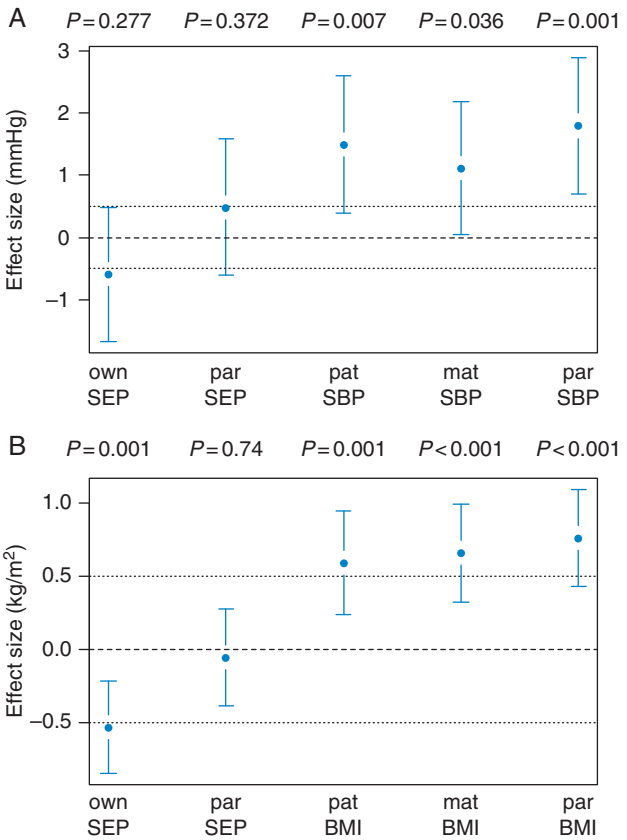

Fig. 1 Comparison of standardized effect sizes (points) and 95% confidence intervals (bars) of own SEP and parental SEP (mutually adjusted), and paternal ('pat'), maternal ('mat') and average parental ('par') CV risk factor for SBP (**A**) and BMI (**B**).

effects accompanying migration seem to affect body fat more than other cardiometabolic risk factors.[38] Burke *et al.* detected significant changes in BMI and other CV risk factors in Australians between age 18 and 25,[39] and in particular, the increases in BMI were higher for those living away from the family home, and even higher for those living with a partner (although the authors do not appear to have adjusted specifically for either childhood or current SEP).

Limitations of this study

Our study had some limitations. We used small area SEP as a proxy for individual SEP measurements, and assumed that each individual has an SEP close to the average for their area. Although this has been shown to be a reasonable assumption,[17,18] particularly when very small areas are used to obtain the aggregate level measure as has been done here with the use of CCDs including ~225 households, the approach may result in non-differential misclassification, as area-level SEP may not match individuals' SEP for some individuals.[40] We would expect that if using area-level SEP as a proxy for individual SEP were to have an effect it would be most likely to bias results towards the null. We also assumed that current parental SEP is equal to childhood SEP for the young adults in this study, and that all siblings and parents within a family shared a household throughout each individual's childhood. If these assumptions were not met, results may be biased towards the null. The length of time spent living away from one's parents, the reason for moving away from the family home (for example, study, work or marriage), whether individuals are working or studying (and if studying, their housing choice), may also be relevant but this information was not available.

Conclusion

In this first study of SEP and CV risk in young Australian adult siblings, we have shown that a decrease in SEP is associated with higher BMI, after adjusting for shared family factors. This is important as the aetiology of CV risk factors including blood pressure and BMI remains somewhat unclear, consequently, elucidating the contribution of genes and environmental factors is important for development of targeted interventions.

Supplementary data

Supplementary data are available at *PUBMED* online.

Acknowledgements

We thank Margaret Stebbing, the general practitioners and research nurses, John Hopper, and Professor Graham G. Giles

and the Health 2000 Study for their contributions to subject recruitment.

Funding

This research was facilitated through the Australian Twin Registry, a national resource in part supported by a Centre of Research Excellence Grant from the National Health and Medical Research Council. This work was supported by the Victorian Health Promotion Foundation and the National Health and Medical Research Council of Australia.

References

1 Kavanagh A, Bentley RJ, Turrell G *et al*. Socioeconomic position, gender, health behaviours and biomarkers of cardiovascular disease and diabetes. *Soc Sci Med* 2010;**71**:1150–60.

2 Myllykangas M, Pekkanen J, Rasi V *et al*. Haemostatic and other cardiovascular risk factors, and socioeconomic status among middle-aged Finnish men and women. *Int J Epidemiol* 1995;**24**:1110–6.

3 Williams ED, Magliano DJ, Zimmet PZ *et al*. Area-level socioeconomic status and incidence of abnormal glucose metabolism the Australian diabetes, obesity and lifestyle (AusDiab) study. *Diabetes Care* 2012;**35**:1455–61.

4 Diez Roux AV. Residential environments and cardiovascular risk. *J Urban Health* 2003;**80**:569–89.

5 Chow CK, Lock K, Teo K *et al*. Environmental and societal influences acting on cardiovascular risk factors and disease at a population level: a review. *Int J Epidemiol* 2009;**38**:1580–94.

6 Diez-Roux AV. Persistent social patterning of cardiovascular risk—Rethinking the familiar. *Circulation* 2005;**111**:3020–1.

7 Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Ann Epidemiol* 2006;**16**:91–104.

8 Laitinen TT, Pahkala K, Venn A *et al*. Childhood lifestyle and clinical determinants of adult ideal cardiovascular health. The Cardiovascular Risk in Young Finns Study, the Childhood Determinants of Adult Health Study, the Princeton Follow-up Study. *Int J Cardiol* 2013;**169**:126–32.

9 Lawlor DA, Ebrahim S, Smith GD. Socioeconomic position in childhood and adulthood and insulin resistance: cross sectional survey using data from British women's heart and health study. *Br Med J* 2002;**325**:805–7.

10 Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. *BMC Public Health* 2005;**5**:7.

11 d'Addio A. Intergenerational transmission of disadvantage: mobility or immobility across generations? In: OECD Social, Employment and Migration Working Papers, OECD Publishing, 2007.

12 Harrap SB, Stebbing M, Hopper JL *et al*. Familial patterns of covariation for cardiovascular risk factors in adults: the Victorian Family Heart Study. *Am J Epidemiol* 2000;**152**:704–15.

13 Cui J, Hopper JL, Harrap SB. Antihypertensive treatments obscure familial contributions to blood pressure variation. *Hypertension* 2003;**41**:207–10.

14 Tobin MD, Sheehan NA, Scurrah KJ *et al*. Adjusting for treatment effects in studies of quantitative traits: antihypertensive therapy and systolic blood pressure. *Stat Med* 2005;**24**:2911–35.

15 Australian Bureau of Statistics. *1996 Census of Population and Housing: Fact Sheet 2—Number of 1996 Census Geographic Areas*. Canberra: ABS, 1999.

16 Australian Bureau of Statistics. *1996 Census Dictionary*. Canberra: ABS, 1996.

17 Australian Bureau of Statistics. *1996 Census of Population and Housing: Socio-economic Indexes for Areas*. Canberra: ABS, 1998.

18 Kavanagh AM, Goller JL, King T *et al*. Urban area disadvantage and physical activity: a multilevel study in Melbourne, Australia. *J Epidemiol Community Health* 2005;**59**:934–40.

19 Institute ESR. ArcGIS (v 9.1). Redlands CA2005.

20 Thornton L, Pearce J, Kavanagh A. Using Geographic Information Systems (GIS) to assess the role of the built environment in influencing obesity: a glossary. *Int J Behav Nutr Phys Act* 2011;**8**:71.

21 Almasy L, Blangero J. Multipoint quantitative-trait linkage analysis in general pedigrees. *Am J Hum Genet* 1998;**62**:1198–211.

22 Fujiwara T, Kawachi I. Is education causally related to better health? A twin fixed-effect study in the USA. *Int J Epidemiol* 2009;**38**:1310–22.

23 Krieger N, Chen JT, Coull BA *et al*. Lifetime socioeconomic position and twins' health: An analysis of 308 pairs of United States women twins. *PLOS Med* 2005;**2**:645–53.

24 Fletcher JM. The effects of first occupation on long term health status: evidence from the Wisconsin longitudinal study. *J Lab Res* 2012;**33**:49–75.

25 Lundborg P. The health returns to schooling-what can we learn from twins? *J Popul Econ* 2013;**26**:673–701.

26 Behrman JR, Kohler HP, Jensen VM *et al*. Does more schooling reduce hospitalization and delay mortality? New evidence based on Danish twins. *Demography* 2011;**48**:1347–75.

27 Hogberg L, Cnattingius S, Lundholm C *et al*. Intergenerational social mobility and the risk of hypertension. *J Epidemiol Community Health* 2012;**66**:e9.

28 Naess O, Hoff DA, Lawlor D *et al*. Education and adult cause-specific mortality-examining the impact of family factors shared by 871 367 Norwegian siblings. *Int J Epidemiol* 2012;**41**:1683–91.

29 Tiikkaja S, Olsson M, Malki N *et al*. Familial risk of premature cardiovascular mortality and the impact of intergenerational occupational class mobility. *Soc Sci Med* 2012;**75**:1883–90.

30 Osler M, McGue M, Christensen K. Socioeconomic position and twins' health: a life-course analysis of 1266 pairs of middle-aged Danish twins. *Int J Epidemiol* 2007;**36**:77–83.

31 Amin V, Behrman JR, Spector TD. Does more schooling improve health outcomes and health related behaviors? Evidence from UK twins. *Econ Educ Rev* 2013;**35**:134–48.

32 Behrman J, Xiong Y, Zhang JS. Schooling-Health Associations Misrepresent Causal Schooling Effects on Health: Evidence from Chinese Adult Twins. *Soc Sci Med* 2015;**127**:190–97.

33 Sovio U, Giambartolomei C, Kinra S *et al*. Early and current socio-economic position and cardiometabolic risk factors in the Indian Migration Study. *Eur J Prev Cardiol* 2013;**20**:844–53.

34 Webbink D, Martin NG, Visscher PM. Does education reduce the probability of being overweight? *J Health Econ* 2010;**29**:29–38.

35 Loucks EB, Buka SL, Rogers ML *et al*. Education and coronary heart disease risk associations may be affected by early-life common prior causes: a propensity matching analysis. *Ann Epidemiol* 2012;**22**: 221–32.

36 Sondergaard G, Mortensen LH, Andersen AMN *et al*. Does shared family background influence the impact of educational differences on early mortality? *Am J Epidemiol* 2012;**176**:675–83.

37 Shaw RJ, Green MJ, Popham F *et al*. Differences in adiposity trajectories by birth cohort and childhood social class: evidence from cohorts born in the 1930s, 1950s and 1970s in the west of Scotland. *J Epidemiol Community Health* 2014;**68**:550–6.

38 Kinra S, Andersen E, Ben-Shlomo Y *et al*. Association between urban life-years and cardiometabolic risk the Indian migration study. *Am J Epidemiol* 2011;**174**:154–64.

39 Burke V, Beilin LJ, Dunbar D *et al*. Changes in health-related behaviours and cardiovascular risk factors in young adults: associations with living with a partner. *Prev Med* 2004;**39**:722–30.

40 Australian Bureau of Statistics. *Socio-Economic Indexes for Areas: Getting a Handle on Individual Diversity within Areas*. Canberra: ABS, 2011.