North-South Gradients in Britain for Stroke and CHD
Are They Explained by the Same Factors?
R.W. Morris, PhD; P.H. Whincup, FRCP; J.R. Emberson, MSc; F.C. Lampe, MSc; M. Walker, MA; A.G. Shaper, FRCP

Background and Purpose—The geographic variation in CHD and stroke within Great Britain is well known. We aimed to quantify these variations and to determine the contribution of established risk factors.

Methods—This prospective study consisted of 7735 men 40 to 59 years of age in 24 British towns who were followed up for 20 years from screening in 1978 to 1980. We compared age-adjusted incidences of major stroke and CHD events in southern England and the rest of Britain before and after adjustment for established cardiovascular risk factors.

Results—At least 1 episode of stroke occurred in 467 men (3.54 per 1000 person-years, age standardized) and of CHD in 1299 men (10.05 per 1000 person-years). Event rates varied between towns, from 2.00 to 5.45 per 1000 person-years for stroke and from 6.16 to 12.21 per 1000 person-years for CHD. Incidence for both diseases was highest in Scottish towns and lowest in southern English towns (“north-south gradient”). The hazard ratio for stroke in the rest of Britain compared with southern England was 1.44 (95% confidence interval [CI], 1.16 to 1.78); for CHD, it was 1.32 (95% CI, 1.14 to 1.53). After adjustment for baseline systolic blood pressure, smoking status, physical activity, social class, and height, the hazard ratio was 1.24 (95% CI, 1.00 to 1.54) for stroke and 1.17 (95% CI, 1.02 to 1.35) for CHD.

Conclusions—Similar north-south gradients were observed for major stroke and major CHD events. The magnitude of these gradients was considerably diminished when individual risk variables were taken into account. (Stroke. 2003;34: 2604-2611.)

Key Words: cerebrovascular accident ■ cohort studies ■ confounding factors (epidemiology) ■ coronary heart disease ■ geography ■ incidence

Geographic variations in coronary heart disease (CHD) and stroke death rates have long been observed in Britain, with lower mortality in the south of England and higher mortality in the north of England and Scotland. Attempts to explain these variations have usually consisted of analyzing aggregated data on suspected risk factors over geographical areas. When such data for risk factors are analyzed in relation to disease rates, the relationships may be overestimated because of the ecological fallacy. In contrast, the British Regional Heart Study (BRHS) has obtained individual data on risk factors for 7735 individual men in 24 British towns who were followed up for 20 years. It is thus possible to examine differences in disease rates between towns in relation to risk factors measured at an individual level.

The BRHS has now followed up these men for >20 years. We have already demonstrated the geographical variation in incidence of CHD over the first 15 years of follow-up, and have shown that 77% of this variation may be explained by 5 established risk factors: smoking, systolic blood pressure, physical activity, social class, and height.

Methods

The design of the BRHS has been described in detail. In the main phase of the BRHS, 24 towns were selected to represent the range of CHD mortality rates and to include all the major regions of Great Britain. Random samples of ~400 men 40 to 59 years of age drawn from a single general practice in each town were invited for screening. The general practice was chosen to be representative of the socioeconomic composition of the town. A 78% response rate was obtained, and 7735 men were screened between 1978 and 1980.

Physical Measurements

A single team of 3 trained research nurses visited all towns in succession. Towns in close proximity were visited at different times of year. The London School of Hygiene and Tropical Medicine sphygmomanometer was used to measure blood pressure twice in

Received March 6, 2003; final revision received June 26, 2003; accepted July 8, 2003.

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Stroke is available at http://www.strokeaha.org

DOI: 10.1161/01.STR.0000092489.98235.1D
succession. The mean of the 2 readings was used in analyses, with adjustment for observer variation within each town. Height was measured to the nearest millimeter in subjects without shoes and weight to the nearest 0.1 kg in subjects wearing trousers and socks.

**Questionnaire Data**

The research nurses administered a standard questionnaire that included questions on smoking habits, physical activity, and social class based on the longest-held occupation. Smoking was defined as 1 of 5 categories: never smokers, ex-smokers, and smokers of 1 to 19, 20, or ≥21 cigarettes per day. For physical activity, an established 6-category classification was used but was reduced to 4 categories for this analysis: none, occasional, light, and moderate or more (active). Social class was defined by use of the Registrar General’s Classification of Occupations and concerned the longest-held job. Seven categories were defined: I, II, III nonmanual, III manual, IV, V, and armed forces. History of diagnosed CHD or stroke was defined as subject recall of ever having had a doctor’s diagnosis of “angina,” “heart attack,” “myocardial infarction,” or “coronary thrombosis” for CHD and “stroke” for stroke.

**Follow-Up**

All men have been followed up for major nonfatal and fatal CHD events (myocardial infarction and sudden cardiac death) and major fatal and nonfatal stroke. Deaths have been flagged through the National Health Service Central Registers in Southport for England and Wales and in Edinburgh for Scotland. Fatal events resulting from CHD or stroke were recorded if the International Classification of Diseases, ninth revision, codes were 410 to 414 and 430 to 438, respectively. Regular reviews of general practice records have been carried out biennially throughout the follow-up period. Nonfatal myocardial infarctions were defined according to standard criteria. Death and stroke event rates have been >99%. A cross-check between medical records and subjects’ recall of diagnoses confirmed that 97% of CHD diagnoses and 77% of stroke diagnoses were correctly identified by medical records. Also, only 5% of strokes and <1% of CHD events initially identified by medical records were false-positives. The database was updated accordingly. A record review carried out in 2000 completed at least 20 years of follow-up for every subject. We have therefore calculated event rates for all 24 towns over a 20-year follow-up period for both CHD and stroke events.

**Statistical Methods**

Event rates for first CHD or stroke were calculated per 1000 person-years; follow-up time was counted as the elapsed years between screening and the first event for men who experienced the event and as 20 years for other men unless they died of other causes before 20 years, in which case they were censored at that time. For each town, these rates were calculated within the age groups of 40 to 44, 45 to 49, 50 to 54, and 55 to 59 years and then averaged to obtain an age-standardized event rate.

Five risk factors (smoking, systolic blood pressure, physical activity, social class, and height) were selected. These factors had explained 77% of the variation in incidence of CHD over a 15-year follow-up in the BRHS. The first 2 have well-established relationships with both CHD and stroke. The latter 3 have been demonstrated to be related to either CHD or stroke in the BRHS and other studies. Serum total cholesterol, although a classic risk factor for CHD, is not related to stroke incidence, and our previous work found that it failed to explain geographical variation in CHD. Height was taken as a proxy marker for deprivation in various life stages before adulthood, and social class was a marker for social disadvantage in adult life.

Pearson correlations of age-standardized CHD and stroke incidence with mean levels of systolic blood pressure and height and prevalence of current cigarette smoking, moderate or vigorous physical activity, and manual social class were calculated for the 24 towns.

**Multilevel Modeling**

Because a sample of 24 towns was chosen from a larger number of possible towns and because subjects were chosen from each of the towns, the data formed a multilevel structure. The statistical package MLwiN was used, including a macro for fitting Cox’s proportional-hazards model for survival data grouped into years of follow-up. All models were adjusted for subject age as a continuous variable and included region. The 2 chief subject-level variables (smoking status, systolic blood pressure) were entered. Then, physical activity, social class, and height were added, and the residual between-town variance was noted.

Each town was classified according to whether it was in southern England or the rest of Britain. This dichotomous classification was included in our models as a town-level variable rather than an individual-level variable. Hazard ratios were calculated for the rest of Britain compared with the south of England.

**Results**

Over 20 years of follow-up, major CHD events occurred in 1299 of 7735 men (16.8%), equivalent to 10.05 first events per 1000 person-years of follow-up. Stroke events occurred in 467 men (6.0%, 3.54 first events per 1000 person-years). The average age at first stroke was 65.1 years and at first CHD event was 62.3 years. For 97 men, both a CHD event and a stroke event were recorded.

**Association Between Incidences for 2 End Points in 24 Towns**

Table 1 shows the age-standardized 20-year event rates of major CHD and stroke in the 24 towns ordered by region. CHD event rates varied from 6.16 per 1000 person-years in Guildford to 12.21 per 1000 person-years in Dewsbury, whereas stroke event rates varied from 2.00 per 1000 person-years in Guildford to 5.45 per 1000 person-years in Falkirk. There was a correlation of 0.49 between the rates for the 2 diseases (the Figure). Table 1 also shows risk factor distributions for each town. For CHD incidence, strong correlations were observed with the baseline prevalence of current smoking (positively) and mean height (negatively), whereas moderate positive correlations existed with mean systolic blood pressure and prevalence of manual social class. For stroke incidence, moderate correlations were observed with baseline prevalence of current smoking and manual social class (positively) and mean height (negatively). A weak positive correlation existed with mean systolic blood pressure, and no correlation existed with physical activity.

**Role of Individual Risk Factors**

Complete data on smoking, blood pressure, physical activity, social class, and height were available for 7609 men. Table 2 shows hazard ratios for each individual risk factor for the 2 diseases after adjustment for each other and age. Strong associations were found with smoking and blood pressure for both CHD and stroke, with physical activity and height for CHD, and with social class for stroke. Only weak, nonsignificant associations with social class for CHD and with physical activity and height for stroke were noted.

Tables 3 and 4 show the hazard ratios for men living in the rest of Britain compared with southern England for CHD and stroke, respectively. After adjustment only for age, the hazard ratio for the rest of Britain compared with the south of England was 1.32 (95% confidence interval [CI], 1.14 to 1.53) for CHD.
and 1.44 (95% CI, 1.16 to 1.78) for stroke. When smoking and blood pressure were included in the model, the magnitude of the log (hazard ratio) was reduced by 29% for CHD and by 28% for stroke. After additional adjustment for physical activity, social class, and height, the magnitude was reduced by 42% and 40% for CHD and stroke, respectively. For CHD, smoking and systolic blood pressure were the 2 variables most effective in reducing the magnitude of the regional difference (although the other 3 variables were almost as effective), whereas for stroke, systolic blood pressure and social class were most effective for reducing this difference.

Exclusion of Subjects With Prior Diagnosis of CHD or Stroke
At the initial screening, 322 of the 7609 men recalled a doctor diagnosis of CHD or stroke, and for another 6, information on recall of these diagnoses was not available. The analysis was repeated for each end point including only those 7281 subjects who did not recall a doctor diagnosis of CHD or stroke. For stroke, the results were very similar to those obtained before exclusion of the 328 subjects. Hazard ratios for living outside the south of England were 1.46 (95% CI, 1.23 to 1.68; \( P = 0.001 \)) and 1.27 (95% CI, 1.04 to 1.50; \( P = 0.037 \)) before and after adjustment for the 5 variables, and the magnitude of the log (hazard ratio) was reduced by 36% when the 5 variables were included in the model. For CHD, the hazard ratios were somewhat reduced when the 328 subjects were omitted. The hazard ratio was 1.25 (95% CI, 1.10 to 1.40; \( P = 0.003 \)) when adjusted only for age. This was reduced to 1.10 (95% CI, 0.96 to 1.24; \( P = 0.18 \)) when the 5 variables were included. The magnitude of the log (odds ratio) was reduced by 59%.

Discussion

Main Findings
The present analysis has shown that the incidence of stroke was increased in regions of Britain outside the south of England, with hazard ratios of 1.43 (95% CI, 1.20 to 1.71) for CHD and 1.46 (95% CI, 1.23 to 1.68) for stroke. When smoking and blood pressure were included in the model, the magnitude of the log (hazard ratio) was reduced by 29% for CHD and by 28% for stroke. After additional adjustment for physical activity, social class, and height, the magnitude was reduced by 42% and 40% for CHD and stroke, respectively. For CHD, smoking and systolic blood pressure were the 2 variables most effective in reducing the magnitude of the regional difference (although the other 3 variables were almost as effective), whereas for stroke, systolic blood pressure and social class were most effective for reducing this difference.

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England, just as for CHD. The increased incidence outside the south of England was slightly greater for stroke both before and after adjustments for individual risk factors and after exclusion of subjects who recalled a doctor diagnosis of CHD or stroke at baseline. Generally, towns with a high incidence of 1 disease also had high incidence for the other. However, only 97 men experienced both types of disease over the 20-year follow-up; this amounted to about one fifth of the strokes and 1 in 13 of the CHD events. Lower mean blood pressure was the variable that most accounted for the decreased incidence of stroke in the south (18%), whereas lower mean blood pressure and lower smoking prevalence accounted to a similar extent for the decreased incidence of CHD (15% and 14%, respectively). Physical activity and height made very little contribution to explaining the geographic variation in stroke, but their contribution, and that of social class, was almost as great as for smoking and blood pressure in explaining variations in CHD.

Unadjusted hazard ratios for the 2 diseases were 1.32 and 1.44 for men living in the rest of Britain compared with the south of England or, inverting, 0.76 and 0.69 for those living in the south of England compared with the rest of Britain. This suggests the potential to avoid about one quarter of CHD and one third of stroke events occurring in the rest of Britain if conditions enjoyed by men in the south of England could be replicated elsewhere. Decreases in rates of smoking and levels of blood pressure to levels prevalent in the south would reduce the inequality by almost one third.

### Strengths and Weaknesses of the Present Study

The BRHS represents towns in all major regions of Britain. The cardiovascular mortality for these towns differed markedly when the men were screened. Other major British prospective studies of CHD or stroke are based on a restricted geographical location. The known geographic variation in disease rates potentially allowed the study to explore the contribution of known risk factors that may differ in their geographic distribution. Thus, the potential benefit when risk factors are modified at the population level may be estimated. Other studies have examined geographic variation by use of aggregated data. Overall rates of disease have been calculated from routinely collected statistics and related to prevalence of risk factors in different areas. In contrast, the BRHS has been able to avoid possible ecological biases inherent in aggregated data by relating relationships at the individual level to variations in incidence at the town level through multilevel modeling.

In using stroke data ascertained from death certificates (for fatal strokes) and general practice medical records (for nonfatal strokes), we have been unable to distinguish between ischemic and hemorrhagic strokes. It has been suggested that height may be more strongly related to hemorrhagic strokes than ischemic strokes. However, the vast majority of strokes...
of unknown origin are probably ischemic in older British populations.19

The ecological correlations observed between towns for risk factors with stroke events were lower than with CHD, probably because the lower stroke event rates led to incidence estimates that were more liable to sampling variation. The real strength of our analysis was in quantifying the geographic gradient within a multilevel model.

It has been shown that associations between individual risk variables and outcome may be underestimated by taking baseline measures to represent usual levels over the follow-up period.20 An analysis that could account for such imprecision may explain the regional differences even more fully than demonstrated by these findings. However, the use of baseline measurements themselves would be more relevant for prediction of regional differences.

Comparison With Findings of Other Studies

Other studies have pointed out trends in risk factor distribution among areas where stroke mortality was known to differ. Hypertension prevalence and glucose intolerance mirrored a gradient observed for stroke mortality in 3 areas in the United States.21 Among men in 22 Scottish health districts, hospital admission rates for stroke were associated with prevalence of hypertension, alcohol consumption, and absence of fruit consumption.22 Common to both is the importance of raised blood pressure. Our study has shown that this single factor was the strongest explanation for the variation in stroke rates between towns.

Occupational social class was the second-most-effective variable in explaining geographic variation. The relationship has previously been demonstrated at an individual level both in the BRHS cohort and in a Scottish cohort (Renfrew/Paisley study), but in both cases, its effect has been at least partially explained by other risk factors.23 Because height has not explained geographic variation in stroke rates in the present study, the contribution of adult social class may reflect exposures to risk factors acting in adult life that occur more commonly outside the south of England.

This study was limited to British middle-aged men, but a recently mounted study of British women 60 to 79 years of age drawn from almost the same general practices demonstrated similar findings using cross-sectional data on geographical variation in cardiovascular disease prevalence.24

TABLE 3. Comparison of British Regions With South of England for Incidence of CHD After Adjustment for Explanatory Variables Specified*

<table>
<thead>
<tr>
<th>Variables Included</th>
<th>Loge Hazard Ratio (SE)</th>
<th>Reduction in Regional Effect, † %</th>
<th>Hazard Ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0.275 (0.075)</td>
<td>0</td>
<td>1.32 (1.14–1.53)</td>
<td>0.0002</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.236 (0.070)</td>
<td>14</td>
<td>1.27 (1.10–1.41)</td>
<td>0.0007</td>
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<tr>
<td>Blood pressure</td>
<td>0.234 (0.074)</td>
<td>15</td>
<td>1.26 (1.09–1.46)</td>
<td>0.0016</td>
</tr>
<tr>
<td>Physical activity</td>
<td>0.246 (0.072)</td>
<td>11</td>
<td>1.28 (1.11–1.47)</td>
<td>0.0006</td>
</tr>
<tr>
<td>Occupational social class</td>
<td>0.247 (0.070)</td>
<td>10</td>
<td>1.28 (1.12–1.47)</td>
<td>0.0004</td>
</tr>
<tr>
<td>Height</td>
<td>0.249 (0.072)</td>
<td>9</td>
<td>1.28 (1.11–1.48)</td>
<td>0.0005</td>
</tr>
<tr>
<td>Smoking, blood pressure</td>
<td>0.194 (0.071)</td>
<td>29</td>
<td>1.21 (1.05–1.40)</td>
<td>0.006</td>
</tr>
<tr>
<td>Smoking, blood pressure, physical activity</td>
<td>0.159 (0.071)</td>
<td>42</td>
<td>1.17 (1.02–1.35)</td>
<td>0.025</td>
</tr>
</tbody>
</table>

*All models adjust for age.
†Taken as percentage of loge (hazard ratio) for model with no variables except age included.

TABLE 4. Comparison of British Regions With South of England for Incidence of Stroke After Adjustment for Explanatory Variables Specified*

<table>
<thead>
<tr>
<th>Variables Included</th>
<th>Loge Hazard Ratio (SE)</th>
<th>Reduction in Regional Effect, † %</th>
<th>Hazard Ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0.364 (0.109)</td>
<td>0</td>
<td>1.44 (1.16–1.78)</td>
<td>0.0008</td>
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<td>Smoking</td>
<td>0.333 (0.109)</td>
<td>9</td>
<td>1.40 (1.13–1.73)</td>
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<tr>
<td>Blood pressure</td>
<td>0.297 (0.109)</td>
<td>18</td>
<td>1.35 (1.09–1.67)</td>
<td>0.006</td>
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<tr>
<td>Physical activity</td>
<td>0.344 (0.109)</td>
<td>5</td>
<td>1.41 (1.38–1.75)</td>
<td>0.002</td>
</tr>
<tr>
<td>Occupational social class</td>
<td>0.308 (0.110)</td>
<td>15</td>
<td>1.36 (1.09–1.69)</td>
<td>0.005</td>
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<tr>
<td>Height</td>
<td>0.353 (0.109)</td>
<td>3</td>
<td>1.42 (1.15–1.76)</td>
<td>0.001</td>
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<td>Smoking, blood pressure</td>
<td>0.263 (0.110)</td>
<td>28</td>
<td>1.30 (1.05–1.61)</td>
<td>0.017</td>
</tr>
<tr>
<td>Smoking, blood pressure, physical activity</td>
<td>0.219 (0.110)</td>
<td>40</td>
<td>1.24 (1.00–1.54)</td>
<td>0.046</td>
</tr>
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</table>

*All models adjust for age.
†Taken as percentage of loge (hazard ratio) for model with no variables except age included.
Public Health Implications

Although differences in the prevalence of the established risk factors appear to make a substantial contribution to the modest increase in risk of stroke and CHD associated with living beyond southern England, it should be recognized that mean levels of serum total cholesterol and body mass index are uniformly high throughout Britain and that the prevalence rates of other risk factors even in southern England are far from desirable. The relatively small changes in blood pressure, cigarette smoking, and physical activity required to bring the rest of Britain down to the levels of CHD and stroke encountered in southern England cannot be seen as a major public health objective when the levels of cardiovascular risk in southern England are still high by international standards. Clearly, any public health actions on diet, body weight, smoking, physical activity, and control of blood pressure must be directed to the whole population throughout Britain and not targeted at specific geographical groups.

Acknowledgments

The BRHS is funded by the British Heart Foundation with additional support from the Department of Health. Opinions expressed in the article are those of the authors and not necessarily those of the funding bodies.

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Editorial Comment

North, South: Changing Directions in Cardiovascular Epidemiology

Despite impressive declines in cardiovascular disease (CVD) over the last half-century, stroke and coronary heart disease (CHD) still constitute the greatest disease burden in the developed world. Moreover, there is accumulating evidence that developing countries will be faced with stroke and CHD epidemics in the relatively near future.¹ Much of our understanding of the etiology of CVD has been gained from prospective cohort studies such as the British Regional Heart Study (BRHS), and in this issue of Stroke, Morris and colleagues² supplement a previous report from this study of geographical patterns of CHD incidence³ by extending the length of follow-up and examining geographical variations in stroke. The BRHS is an ideal study for investigating the geographical inequalities in CVD. Indeed, the study was established with this aim in mind, specifically to test the hypothesis that water quality was a determinant of CVD risk, which proved not to be the case.⁴ However, despite the fact that the study generated >250 articles, mostly on the causes
The relevance of this issue is that if variations in health and consequences of CVD, it was not until 2001 (>20 years after the initiation of the study) that the “definitive paper about the causes of regional variation in coronary heart disease appeared.” This most recent contribution is therefore welcome. The authors found that among men the risks of both CHD and stroke were greater in the rest of Britain compared with the south of England and that this difference was substantially, although not completely, explained by adjustment for a number of adult CVD risk factors: systolic blood pressure, smoking status, physical activity, social class, and height. Had they adjusted for other established risk factors, in particular diabetes status and dyslipidemia, which are associated with both CHD and occlusive stroke, the residual variation may have been completely removed. These findings are in line with those of a similar previous study of British men. What then does this work add to our epidemiological and public health knowledge? In part, the answer to this question requires a greater understanding of the factors responsible for geographical variations in CVD risk factors and thus CVD.

The results essentially confirm the association between several established adult risk factors and CVD, and the authors acknowledge in their concluding paragraphs that their findings have little to contribute to public health practice: “Clearly, any public health actions on diet . . . must be directed to the whole population throughout Britain and not targeted to specific geographically located groups.” The article refers to the north-south gradient, but in fact the comparisons are dichotomized between the south of England and the rest of Britain. From Table 1, it can be seen that in this particular study there is not a clear north-south gradient: Merthyr Tydfil, Gloucester, and Shrewsbury, all of which have southern latitudes, have high incidences of both CHD and stroke, whereas Darlington in the north has a relatively low risk of CHD, and Harrogate, also in the north, has a relatively low risk of stroke. This reflects in part the selection of towns in the BRHS, and although a north-south gradient was not observed, there is substantial geographical variation, with incidences of CHD varying between 6.16 and 12.21/1000 person-years and of stroke between 2.00 and 5.45/1000 person-years across the 24 towns. The between-town variation in both CVD and risk factor occurrence is likely to be explained by area- and/or individual- level deprivation; the authors might therefore have explored this geographic diversity within the BRHS and sought explanations beyond established adult risk factors.

Many studies have demonstrated geographical inequalities of the sort presented here. Importantly, many investigators have now moved beyond these simple ecological designs and examined individual- and area-level measures to determine whether the physical and social aspects of where people live influence health independently of the characteristics of the people themselves. The relevance of this issue is that if variations in health between areas can be entirely explained by the personal characteristics of the inhabitants of these areas, then policy makers need act only on improving the circumstances of individuals. Conversely, the demonstration of independent area-level effects would be key in emphasizing the need to focus attention on features of the areas where people live and not just the individuals living there. This is important because the widening gap between the rich and poor appears to be mirrored by a growing divergence of their residential environments, so that affluent people are increasingly living and interacting with other affluent people while the poor increasingly live and interact with other poor people. Such studies could go even further; rather than simply use census-derived contextual effects, the environments in which study participants live could and should be examined. Both the BRHS and the newly formed British Women’s Heart and Health Study could in the future incorporate an examination of the neighborhoods in which their participants live by assessing, for example, the following: the local availability of affordable fruits and vegetables, green areas, and physical activity facilities; area levels of criminal activity; and other indicators of environmental adversity.

That the baseline measurements in this study were taken 20 years before the final period of follow-up reaffirms the need to combat adverse risk factor profiles at least as early as middle age. However, the findings also prompt a wider consideration of cardiovascular risk. Blood pressure in middle age may be a strong risk factor but is itself set in train in early life, as evidenced by the declines seen in several regions over the last 50 years in young people who were not taking antihypertensive medication. Height is also a measure of early life exposures, and its association with CVD (demonstrated in the BRHS and other studies) may represent the role of genes, early nutrition or infection, or other socioeconomic exposures that become embodied over the years. It has been claimed that the major risk factors (smoking, dyslipidemia, hypertension, inactivity) for CVD are known and that emphasis should now be placed on tackling these rather than searching for other risk factors. However, these risk factors do not explain socioeconomic variations in CVD and are themselves determined by social, environmental, and biological exposures acting throughout the course of life.

Finally, it is opportune to consider the growing burden of CVD in developed countries and the potential for it to greatly widen the global north-south gradient in health. With respect to exposures over the life course, the greatest risks to public health are likely to be seen in developing countries where the effects of extreme poverty in early life and in earlier generations are being exacerbated through the adoption of adverse Western diets and lifestyles in adulthood. Cardiovascular research is increasingly carried out in developing countries, and continued support for this body of work is required. Similarly, it is imperative that interventions aimed at preserving the cardiovascular health of young individuals in developed countries are also implemented in more deprived parts of the globe. Geography provides a valuable tool for a more comprehensive investigation of disease etiology; it is essential that it is not used merely as an indicator of health inequality.

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Stroke. 2003;34:2604-2609; originally published online October 9, 2003;
doi: 10.1161/01.STR.000092489.98235.1D

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