

supportive counselling achieved an intermediary position between cognitive behaviour therapy and routine care alone, suggesting that non-specific psychological effects—such as intensive interest and support—can have a beneficial effect for patients with chronic psychosis. We tentatively conclude that cognitive behaviour therapy, used as an adjunct treatment for chronic schizophrenia, can result in clinical benefits in the short term.

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Contributors: NT had the original idea for the study, obtained grant funding, and acted as principal investigator. The study protocol was designed by NT, LY, CK, and JM. LY designed the therapy programmes, produced the therapy manuals, and carried out therapy assisted by AG and NT. CK and EMcC were responsible for screening, recruitment, and assessments. Data analyses were carried out by CK, EMcC with advice from JM. GH was responsible for monitoring and assessing treatment fidelity. All authors contributed to the discussion of core ideas. NT had responsibility for writing the paper with contributions from all the other authors.

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Number needed to screen: development of a statistic for disease screening

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Abstract

Objectives: To develop the number needed to screen, a new statistic to overcome inappropriate national strategies for disease screening. Number needed to screen is defined as the number of people that need to be screened for a given duration to prevent one death or adverse event.

Design: Number needed to screen was calculated from clinical trials that directly measured the effect of a screening strategy. From clinical trials that measured treatment benefit, the number needed to screen was estimated as the number needed to treat from the trial divided by the prevalence of heretofore unrecognised or untreated disease. Directly calculated values were then compared with estimate number needed to screen values.

Subjects: Standard literature review.

Results: For prevention of total mortality the most effective screening test was a lipid profile. The estimated number needed to screen for dyslipidaemia (low density lipoprotein cholesterol concentration > 4.14 mmol/l) was 418 if detection was followed by pravastatin treatment for 5 years. This indicates that one death in 5 years could be prevented by screening

418 people. The estimated number needed to screen for hypertension was between 274 and 1307 for 5 years (for 10 mm Hg and 6 mm Hg diastolic blood pressure reduction respectively) if detection was followed by treatment based on a diuretic. Screening with haemocult testing and mammography significantly decreased cancer specific, but not total, mortality. The number needed to screen for haemocult screening to prevent a death from colon cancer was 1374 for 5 years, and the number needed to screen for mammography to prevent a death from breast cancer was 2451 for 5 years for women aged 50-59.

Conclusion: These data allow the clinician to prioritise screening strategies. Of the screening strategies evaluated, screening for, and treatment of, dyslipidaemia and hypertension seem to produce the largest clinical benefit.

Introduction

Too often politics, rather than evidence, dictates the national strategy for disease screening. There are too few clinical trials showing the efficacy of screening strategies.¹⁻⁴ More randomised trials are needed. In the

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meantime a strategy for disease screening based on available evidence is needed.

The ability to compare the efficacy of screening strategies is a prerequisite for the development of a national strategy for disease screening. Until now there has been no means of comparing the overall benefit of screening. The results of most clinical trials are presented as relative risk reduction or odds ratios, but these ignore the role of event rate on overall clinical benefit. For example, when presented as relative risk reduction a highly effective screening strategy for a disease with a low mortality will seem better than a less effective screening strategy for a disease with higher mortality. Furthermore, doctors and patients sometimes interpret the degree of statistical significance as an index of clinical relevance, but this ignores the effect of study size on significance. A modestly effective screening strategy studied in a large number of people can result in a lower P value than that observed with a highly effective screening strategy studied in a smaller number of people.

In clinical trials comparing treatments a better quantitation of overall clinical benefit is provided by presenting results as number needed to treat. Number needed to treat is defined as the number of people that need to be treated for a given duration to prevent one death or one adverse event.^{5,6} Number needed to treat is the reciprocal of the absolute risk reduction. The ideal number needed to treat is 1, indicating that all treated patients will benefit. Less effective treatments have higher values. A positive number indicates that the treatment benefits the patient and a negative number that the patient is harmed by the treatment. Confidence intervals can be calculated. A significant number occurs if the 95% confidence intervals are either both positive or both negative.

I extended the number needed to treat concept to compare strategies for disease screening. I developed a new statistic termed the number needed to screen, defined as the number of people that need to be screened to prevent one death or one adverse event, and calculated number needed to screen values for the prevention of all cause death. I propose that number needed to screen could form the basis of a strategy for disease screening. For some screening methods number needed to screen values were calculated on the basis of the results of screening clinical trials for example, mammography and haemocult. Unfortunately, there are no trials evaluating the prevention of death by screening for atherosclerotic risk factors. I therefore estimated number needed to screen values for atherosclerotic risk factors on the basis of the results of treatment clinical trials and the prevalence of inadequately treated risk factors.

Methods

I identified studies of disease prevention from recent literature reviews, meta-analyses, and Medline searches. If a complete recent meta-analysis, including raw mortality data, was found in the literature, data were collected. Otherwise, original articles were identified and consulted as in a standard meta-analysis. All studies with drugs were randomised and double blind. Other studies were randomised but not blinded for obvious reasons (for example mammography). Meta-

analysis was performed in a cumulative manner as described⁷ with precautions noted.⁸ Absolute risk reduction with 95% confidence intervals were calculated using the random effects model,^{8,9} which produces estimates of interstudy heterogeneity. Heterogeneity is a measure of statistical difference between studies. There was no evidence for heterogeneity by χ^2 analysis in the studies analysed. Significance was defined as $P < 0.05$ with two sided hypothesis testing.

Number needed to treat analysis^{5,6} was performed by calculating the absolute event reduction with 95% confidence intervals for each meta-analysis. Number needed to treat equals 1 divided by absolute risk reduction. In clinical trials that directly tested the benefit of a screening strategy, the number needed to screen was calculated as number needed to screen equals 1 divided by absolute risk reduction.

I included the following screening methods for cancer of the colon and breast: screening haemocult in the prevention of colon cancer and all cause mortality (meta-analysis⁴) and screening mammography in the prevention of breast cancer¹⁻³ and total mortality.^{2,3}

There are no large mortality studies evaluating the benefit of screening for atherosclerotic risk factors. There are, however, several large trials showing the benefit of treating hypertension and dyslipidaemia once these conditions have been detected. To calculate the benefit of screening and then treating atherosclerotic risk factors, knowledge of the benefit of treating these risk factors is needed. I calculated number needed to treat values from the following trials, which evaluated the benefit of treating atherosclerotic risk factors in people without known atherosclerosis: diuretic based treatment of mild hypertension,^{5,10-19} diuretic based treatment of mild hypertension with large decreases in blood pressure,^{10,12,14,19} β blocker based treatment of mild hypertension,^{5,15,20} the antidy-lipidaemic drugs pravastatin,^{21,22} gemfibrozil,²³ clofibrate,²⁴ and aspirin,^{25,26} diet for dyslipidaemia,²⁷ and antidy-lipidaemic bile acid binding resins.^{28,29}

To calculate the number needed to screen from clinical trials that measure the benefit of treating risk factors, a knowledge of the prevalence of disease that can be detected by screening is needed. I obtained estimates of the prevalence of unrecognised and untreated atherosclerotic risk factors from the atherosclerosis risk in communities study.³⁰ These estimates are old; the data were collected between 1987 and 1989.³⁰ Unfortunately, more recent data are not available. Of 15 739 North Americans studied, 2770 (17.6%) had uncontrolled systolic (>140 mm Hg) or diastolic (>90 mm Hg) hypertension, and 4076 (25.9%) had uncontrolled dyslipidaemia, defined as a total cholesterol concentration >6.21 mmol/l. To calculate number needed to screen I assumed that the population with a total cholesterol concentration >6.21 mmol/l was similar to a population with concentrations of low density lipoprotein cholesterol concentrations >4.14 mmol/l, which is similar to the population studied in the treatment trials.²¹⁻²⁹ If the prevalence of low density lipoprotein cholesterol concentration >4.14 mmol/l is less than 27%, I overestimated the benefit of screening for dyslipidaemia.

Number needed to screen was then calculated by dividing the number needed to treat for treating risk

Table 1 Number needed to screen concept

Mortality		Risk reduction		No needed to screen†
Control (%)	Treatment (%)	Relative (%)	Absolute*	
5	4	20	1	100
0.5	0.4	20	0.1	1000
0.05	0.04	20	0.01	10000

*Control mortality × relative risk reduction.

†100 divided by absolute risk reduction.

factors by the prevalence of disease that was unrecognised or untreated. This analysis is subject to propagation of errors because the divisors come from two different studies. Therefore, results must be analysed cautiously. I assumed that screening for hypertension with sphygmomanometry and for dyslipidaemia with laboratory testing identified all patients with disease. This is a reasonable assumption in hypertension and dyslipidaemia considering that appropriately performed sphygmomanometry and laboratory testing define hypertension and dyslipidaemia respectively.

Number needed to screen values were normalised to 5 years to allow comparison between trials with differing durations. This normalisation was appropriate since these trials lasted between 3 and 9 years. The primary endpoint analysed was total mortality because it is least susceptible to post hoc interpretation. In the case of cancer screening, cancer specific mortality was also analysed.

Results

An example of the number needed to screen concept can be shown by screening strategies that decrease

mortality by 20% (a relative risk reduction of 20%, table 1). Firstly, if a disease has a high unscreened mortality of 5%, screening would reduce mortality to 4% (20% of 5%). The absolute risk reduction is 1% (5% minus 4%) and the number needed to screen is 100 (1 divided by 1%). For every 100 unscreened people five will die, and for every 100 screened people only four will die. Screening of 100 people therefore saved one life. If, however, another disease has a lower unscreened mortality of 0.5%, screening would reduce mortality to 0.4% (20% of 0.5%). The absolute risk reduction is 0.1% (0.5% minus 0.4%) and the number needed to screen is 1000 (1 divided by 0.1%). In this case, 1000 people need to be screened to save one life. For a third disease with a very low unscreened mortality of 0.05%, the number needed to screen is even higher. Screening would reduce mortality to 0.04% (20% of 0.05%). The absolute risk reduction is 0.01% (0.05% minus 0.04%) and the number needed to screen is 10 000 (1 divided by 0.01%). A positive number needed to screen implies that screening prevented a death, and a negative number implies that screening increased mortality.

The benefits of screening for cancer of the colon and breast have been tested in large clinical trials. In three trials, screening haemoccult resulted in a number needed to screen of 808 to prevent a death from colon cancer in 8.5 years, a value that was statistically significant (table 2). To prevent a death from breast cancer in 9 years the number needed to screen was 695 for women aged 60-69. Younger women had a higher number needed to screen, as would be expected from the lower prevalence of breast cancer in such people. There was no significant benefit in total mortality in screening for cancer of the colon or breast.

Table 2 Primary prevention of cancer of the breast and colon by screening

	No needed to screen (95% CI)	Duration (years)	No of		Risk reduction (%)	
			Trials	Patients	Relative	Absolute
Cancer specific mortality:						
Screening haemoccult	808 (562 to 1648)*	8.5	3	130 073	23	0.12
Screening mammography:	1887 (1343 to 3505)*	8.5	7	372 612	19	0.05
Age 60-69	695 (474 to 1699)*	9	1	71 444	31	0.14
Age 50-59	1532 (985 to 4782)*	8	2	149 849	23	0.06
Age 40-49	4576 (2001 to -6584)	8.8	2	136 763	13	0.02
Total mortality:						
Screening haemoccult	4894 (253 to -235)	3.1	1	21 757	1	0.02
Screening mammography	-7660 (951 to -672)	7.2	1	89 835	-1.4	0

*Statistically significant, values are not normalised to trial duration (negative number indicates screening increased mortality).

Table 3 Primary prevention of death with cardiovascular agents in patients with no atherosclerotic cardiovascular disease

	No needed to treat (95% CI)	Duration (years)	No of		Risk reduction (%)	
			Trials	Patients	Relative	Absolute
Antihypertensive drugs*:						
Diuretics (decrease 10.0)	43 (26 to 243)†	5.6	4	3 141	18	2.2
Diuretics (decrease 5.7)	213 (136 to 552)†	5.4	11	48 013	8	0.4
β blockers (decrease 6)	332 (18 to -18)	5.2	3	22 729	6	0.3
Antidyslipidaemic drugs:						
Pravastatin	126 (71 to 24035)†	4.3	2	7 657	22	0.7
Diet	85 (42 to -88)	9	1	1 232	31	1.1
Resin	203 (78 to -192)	5.4	2	6 084	13	0.4
Gemofibrozil	-799 (161 to -79)	5	1	4 081	-5	-0.1
Clofibrate	-156 (-5398 to -70)†	5.3	1	10 627	-26	-0.6
Aspirin	340 (149 to -765)	5.2	2	27 212	8	0.2

*In the absence of known atherosclerotic cardiovascular disease. Bracketed data refers to decrease in diastolic blood pressure.

†Statistically significant, values are not normalised to trial duration (negative number needed to treat indicates that screening increased mortality).

Table 4 Comparison of number needed to screen in primary prevention of death

Disease	Prevalence of untreated disease	Screen (treatment)	No needed to screen for 5 years (95% CI)†
Total mortality			
Dyslipidaemia	0.26	Lipid profile (pravastatin)	418 (235 to 79 720)*
		Lipid profile (diet)	590 (292 to -610)
		Lipid profile (resin)	846 (325 to -799)
Hypertension	0.18	Sphygmomanometer (diuretics) (diastolic blood pressure decrease 10)	274 (165 to 1546)*
		Sphygmomanometer (diuretics) (diastolic blood pressure decrease 6)	1307 (834 to 3386)*
		Sphygmomanometer (β blockers)	1961 (106 to -105)
Coronary artery disease		Question (aspirin)	354 (155 to -795)
Colon cancer		Haemoccult (standard)	3034 (157 to -145)
Breast cancer		Mammography (standard)	-11 029 (1369 to -967)
Cancer specific mortality			
Colon cancer		Haemoccult (standard)	1374 (955 to 2802)*
Breast cancer:			
Age 60-69		Mammography (standard)	1251 (853 to 3058)*
Age 50-59		Mammography (standard)	2451 (1576 to 7651)*
Age 40-49		Mammography (standard)	8054 (3522 to -11587)

*Statistically significant.

†Only mortality trial errors taken into account; errors in quit rates not included (negative number indicates screening increased mortality).

There are no large mortality studies evaluating the benefit of screening for atherosclerotic risk factors. There are, however, several large trials showing the benefit of treating hypertension and dyslipidaemia once these conditions have been detected. I developed a method to extrapolate the results of treatment trials to evaluate the benefits of screening. In clinical trials that measure treatment benefit, the number needed to treat from the trial divided by the prevalence of so far unrecognised or untreated disease equals the number needed to screen. For example, in a population of 400 people of whom 125 have a risk factor, if only 25 people are adequately treated, then 100 people (25% of the population) either are unaware of the risk factor or the risk factor is not adequately treated. If clinical trials of treatment showed a number needed to treat of 100 to prevent a death then treating these 100 unaware or untreated people will prevent one death. The number needed to screen is 400 because 400 people would be screened to identify the 100 who need to be treated. This value is calculated as the number needed to treat (100) divided by the prevalence of unaware or untreated people (0.25).

I calculated number needed to treat values for treating atherosclerotic risk factors once they have been identified (table 3). Treatment of hypertension for 5.4 years with regimens containing thiazide diuretic decreased mortality, with a number needed to treat of 213 to prevent one death. If the four studies with the largest reduction in diastolic blood pressure (reduction of 10.0 mm Hg) are analysed, number needed to treat was lower at 43 for 5.6 years, suggesting further benefit for aggressive blood pressure reduction. There was less benefit with antihypertensive regimens mainly containing β blockers (number needed to treat 332 for 5.2 years, not significant). In people without known atherosclerotic vascular disease, pravastatin treatment for dyslipidaemia resulted in a number needed to treat of 126 for 4.3 years. The number needed to treat values

for diet (85 for 9 years) and cholestyramine (203 for 5.4 years) were similar: these values did not reach statistical significance because fewer people were studied. Gemfibrozil had no benefit on total mortality (gemfibrozil did prevent myocardial infarctions, especially in people with high triglyceride concentrations²³). Clofibrate, a drug no longer approved for the treatment of dyslipidaemia, showed a significant increase in total mortality with a number needed to treat of -156 for 5.3 years indicating that one person died for each 156 people treated. Aspirin in healthy men resulted in a number needed to treat of 340 for 5.2 years, a value that did not reach statistical significance.

Number needed to screen values were calculated and then normalised to 5 years (table 4). For prevention of total mortality the most effective screening test was a lipid profile. If screening showed a high low density lipoprotein cholesterol concentration >4.14 mmol/L, treatment with pravastatin for 5 years resulted in a number needed to screen of 418. This value suggests that one death in 5 years could be prevented by screening 418 people. Screening for hypertension also decreased total mortality if detection was followed by treatment based on a diuretic. The number needed to screen values were 274 and 1307 for 10 and 5.7 mm Hg decreases in diastolic blood pressure respectively. None of the other screening strategies had statistically significant effects on total mortality. The estimated number needed to screen values for treating everyone with aspirin or treating dyslipidaemia with diet or cholestyramine were similar to the values for treating with pravastatin, but were not statistically significant. This could represent a type 2 error because fewer people were studied. The number needed to screen values for β blockers in hypertension, haemoccult screening for colon cancer, and mammography for breast cancer were larger than those for dyslipidaemia indicating less possibility for benefit in total mortality. There was a benefit in cancer specific mortality for screening haemoccult and for mammography in women between the ages of 50-69.

Discussion

The major finding of this study is that screening for, and treatment of, dyslipidaemia and hypertension should be a main goal of the healthcare system. Estimated number needed to screen values for dyslipidaemia and hypertension screening were at least fourfold lower than those for screening for cancer of the breast or colon. This suggests that, compared with screening for dyslipidaemia or hypertension, at least four times as many people need to be screened for cancer of the breast or colon to prevent a death.

The major assumption made in this analysis was the estimated prevalence of undiagnosed and untreated hypertension and dyslipidaemia. My estimates were based on a survey conducted from 1987 to 1989. It is likely that the treatment rate of hypertension and dyslipidaemia has improved since 1989. Assuming I overestimated the prevalence of undiagnosed and untreated hypertension and dyslipidaemia by a factor of two, then I would need to correct the number needed to screen values by multiplying them by 2. With this correction, estimated number needed to screen for dyslipidaemia and hypertension screening (836 and

548 to 2614 respectively) would still be twofold lower than the number needed to screen values for screening for cancer of the breast or colon.

This analysis has several additional limitations. Most of these studies of dyslipidaemia and hypertension were performed in middle aged white men. Extrapolation of benefit to women, younger or older people, and non-white people may not be correct. A second limitation is that screening for atherosclerotic risk factors was not explicitly tested in randomised trials. Such trials would be expensive and unethical. The analysis presented in this paper suggests that such trials are not required because the potential benefit is large. Nevertheless, estimated number needed to screen should be evaluated cautiously because division of number needed to treat (table 3) by estimated prevalence is subject to propagation of errors. Thirdly, number needed to screen values may be artefactually low if some of the patients identified by screening decide not to be treated. In practice, compliance with treatments is frequently less than that observed in the clinical trials. Fourthly, for results to apply to patient care the prevalence of disease should be comparable to the population studied. Finally, some benefits may not be linearly related to time so that normalisation of number needed to screen to 5 years may not be appropriate.

These data do not imply that screening for cancer of the breast or colon is inappropriate; clearly screening for these conditions in selected people should continue. The data do suggest that national initiatives should be strengthened to detect and treat dyslipidaemia and hypertension. Despite recent clinical trials showing benefit of treatment, the high prevalence of undiagnosed and untreated hypertension and dyslipidaemia is shameful.

Mammography and haemoccult screening clearly decreased in cancer specific mortality. One reason these results were statistically significant despite higher number needed to screen values was that the mammography and haemoccult studies employed more people. It is possible that these studies underestimate the value of screening for cancer of the breast or colon. Some patients with breast cancer survive for more than 10 years; the benefit from mammography may have been larger if studies were of longer duration. The clinical benefits may also be larger with improved tests for example, biplane mammography or newer cancer treatments. By the same analysis, the clinical benefits of treating hypertension and dyslipidaemia may also be larger with new treatments, for example, angiotensin converting enzyme inhibitors, angiotensin II inhibitors, more potent 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors, or combination treatment. Without clinical trial data extrapolation to newer screening strategies or treatments can be dangerous. Furthermore, new tests and treatments may have adverse effects that are not anticipated, such as class I antiarrhythmics in ischaemic heart disease³¹ or clofibrate in dyslipidaemia.²⁴

This analysis of screening for dyslipidaemia only applies to people who are not known to have atherosclerotic vascular disease. The benefit of treating dyslipidaemia in people with atherosclerosis is much greater than in those without atherosclerosis.⁶ For example, treatment with pravastatin or simvastatin of people with known atherosclerosis decreased total death with a number needed to treat of 37 for 5 years.

Key messages

- Number needed to screen is a new statistic defined as the number of people that need to be screened for a given duration to prevent one death or one adverse event. It can be directly calculated from clinical trials of disease screening, and can also be estimated from clinical trials of treatment and the prevalence of so far unrecognised or untreated disease
- For prevention of all cause death, 418 people need to be screened with a lipid profile if detection of dyslipidaemia was followed by pravastatin treatment for 5 years
- The estimated number needed to screen for hypertension to prevent all cause death was 274 to 1307 for 5 years if detection was followed by treatment with thiazide diuretic
- Screening with haemoccult testing or mammography did not significantly prevent all cause death. Haemoccult screening significantly decreased deaths from colon cancer with a number needed to screen of 1274 for 5 years. Mammography significantly reduced deaths from breast cancer with a number needed to screen of 2451 for 5 years of women aged 50-59

This study showed that screening for, and treatment of, dyslipidaemia and hypertension seem to produce the largest clinical benefit of the screening strategies evaluated.

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Economic change, crime, and mortality crisis in Russia: regional analysis

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Abstract

Objective: To identify which aspects of socioeconomic change were associated with the steep decline in life expectancy in Russia between 1990 and 1994.

Design: Regression analysis of regional data, with percentage fall in male life expectancy as dependent variable and a range of socioeconomic measures reflecting transition, change in income, inequity, and social cohesion as independent variables.

Determination of contribution of deaths from major causes and in each age group to changes in both male and female life expectancy at birth in regions with the smallest and largest declines.

Setting: Regions (oblasts) of European Russia (excluding Siberia and those in the Caucasus affected by the Chechen war).

Subjects: The population of European Russia.

Results: The fall in life expectancy at birth varied widely between regions, with declines for men and women highly correlated. The regions with the largest falls were predominantly urban, with high rates of labour turnover, large increases in recorded crime, and a higher average but unequal distribution of household income. For both men and women increasing rates of death between the ages of 30 and 60 years accounted for most of the fall in life expectancy, with the greatest contributions being from conditions directly or indirectly associated with heavy alcohol consumption.

Conclusions: The decline in life expectancy in Russia in the 1990s cannot be attributed simply to impoverishment. Instead, the impact of social and economic transition, exacerbated by a lack of social cohesion, seems to have played a major part. The evidence that alcohol is an important proximate cause of premature death in Russia is strengthened.

Introduction

The scale of the health crisis facing the Russian people in recent years is now well recognised. After a period of steady improvement after the second world war, life expectancy at birth began to lag behind that in the West in the mid-1960s. A substantial improvement in 1985, coinciding with a major campaign to reduce alcohol consumption,^{1,2} was rapidly reversed and has fallen even further since the collapse of the Soviet Union,³ with life expectancy at birth falling by over 5 years between 1990 and 1994. We have previously shown that these changes cannot be attributed to artefact.⁴

The decline in Russians' life expectancy in the 1990s is clearly driven by profound economic, political, and social changes. There is also considerable evidence that alcohol has played a major part.⁴ The nature of these relations, however, remains unclear. In particular, the relative importance of impoverishment and of the effects of rapid social and economic transition requires elucidation, as some argue that little can be done in the absence of policies to deal with the economic decline that has occurred in the 1990s whereas others have suggested that the effect of rapid change is more important.⁵

The nature of the social and economic transition in Russia has not been uniform, with some regions affected much more than others. There are also large differences in mortality within the regions of Russia.⁶ We explored the nature of the links between economic factors and mortality by taking advantage of this regional diversity. In particular, we examined whether it was possible to distinguish the effects of impoverishment from those resulting from the pace of transition. If the increase in mortality has been a result of impoverishment we would expect the greatest falls in life expectancy in regions experiencing the largest falls in average income; if it has