Spatial and temporal variation of mortality and deprivation 2: statistical modelling

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Abstract. Building on the tabular analyses exemplified in our first paper and widely used in the medical literature, we use generalised linear models to provide a formal, statistical approach to the analysis of mortality and deprivation relationships, and their change over time. Three types of fixed effects model are specified and estimated with the same ward-level data sets for Wales examined in our first paper. They are: Poisson models for analysing mortality and deprivation at a single cross section in time; repeated-measures Poisson models for analysing mortality–deprivation relations, not only at cross sections in time, but also their changes over time; and logit models focusing on temporal changes in mortality–deprivation relationships. Nonlinear effects of deprivation on mortality have been explored by using dummy variables representing deprivation categories to establish the connection between formal statistical models and the tabular approach.

1 Introduction
This is the second of two papers in which we examine the relationship between mortality and material deprivation both over space (wards and ward groups in Wales) and over time (the intercensal period 1981–91). The focus of our first paper (Higgs et al, 1998) was on mortality gradients at cross sections in time and, more especially, their change over time with the ward data aggregated to broad (quintile or decile) groupings ordered on a deprivation scale. In the present paper we show that this style of research, exemplified by the work of Phillimore et al (1994a) and McLoone and Boddy (1994), and relying heavily on nonformal tabular analysis, can be undertaken more systematically with generalised linear models (Aitkin et al, 1989).

This approach allows the relationship between mortality and deprivation to be readily explored, not only with data aggregated to broad deprivation ward groups, but also with the more spatially disaggregated ward data. A wide range of statistical methods is now available for examining the relationship between mortality and deprivation at the small area or individual levels based on the generalised linear model (Aitkin et al, 1989) in both single and multilevel forms (Congdon, 1995; Goldstein, 1995; Gould and Jones, 1996). In this paper, our primary aim is to explore fixed, rather than random, effects approaches to model tabular data commonly used in public health research. In particular, we link the tabular analyses of Phillimore et al (1994a) and McLoone and Boddy (1994) to the Poisson regression modelling of, for example, Shouls et al (1996) and Staines (1994), and extend this research by developing logit and Poisson regression models relating the changing incidence of mortality to cross-sectional deprivation measures. To our knowledge, the logit model has not been applied previously to the study of mortality changes by means of multiple cross-sectional data sets. All these models are estimated with the same data sets for Wales used in our first paper.

The structure of this paper is as follows. In section 2 we provide the statistical framework by discussing Poisson regression models for estimating mortality at the cross section and mortality changes over time. In particular, a logit model for mortality change is developed and compared with a repeated-measures Poisson model. In sections
3 to 6 we then provide empirical applications of the various models by using 800 wards in Wales for which both 1981 and 1991 Census data are available. In section 3 we examine the cross-sectional Poisson models; in section 4 the repeated-measures Poisson model for mortality change; and in section 5 the logit model for mortality change. In section 6 we apply this logit model with the deprivation variable categorised into those quintiles used in our first paper (Higgs et al, 1998).

2 The modelling framework

2.1 General framework

We recall from our first paper that the methodology adopted addresses the two central objectives of determining:

(1) The variation of premature standardised mortality (smr), defined as observed divided by expected deaths, over individuals grouped by ward, j, and subject to different levels of deprivation, \( d_j \), separately at the two cross sections \( t = 1981-83 \) and \( t = 1990-92 \). Formally, this may be expressed as

\[
\text{smr}_j(t) = G_t \left[ d_j \{X(t)\} \right],
\]

in which standardisation is to 1981–83 and 1990–92 separately. The \( d_j \{X(t)\} \) term denotes that deprivation is defined as a function of a set of component variables \( \{X\} \).

(2) Variation in the change in standardised mortality between 1981–83 and 1990–92 over individuals grouped by ward and subject to different levels of material deprivation. Formally

\[
\frac{\Delta \text{smr}_j}{\text{smr}_j(81-83)} = H[d_j\{X(81)\}],
\]

where \( \Delta \text{smr}_j \) is the change in the standardised mortality ratio over the decade as a function of deprivation in 1981. Standardisation is performed with reference to a common time period, here taken as 1981–83 as also done by Phillimore et al (1994a). G and H represent functional dependencies which were assessed through categorical description for selected ranges of deprivation in our first paper (Higgs et al, 1998), and are now treated through formal statistical analysis. The ratio of values of smr in equation (2) formalises the change in mortality used by Phillimore et al (1994a).

To exploit the full range of variability in premature deaths between the wards, we shall explore specific parametric forms for the functions G and H in equations (1) and (2), which are amenable to statistical estimation using generalised linear models (Aitkin et al, 1989). This form of analysis has been undertaken at the cross section and over time by, among others, Staines (1994) and Shouls et al (1996). Compared with the nonformal analysis of our first paper (Higgs et al, 1998) it allows fuller understanding of the association between mortality and deprivation and a framework for exploring the statistical significance of different forms of dependency. The precise specification of the relationship between mortality and deprivation will depend on the selection of explanatory variables in \( \{X\} \), and the constraints imposed on the estimates of deaths in each ward.

2.2 Modelling mortality at the cross section

The deaths in each ward, which are in the form of a count \( D_j \), are treated as independent Poisson variables. The variation in the number of deaths over the \( N \) wards is explained by a set of independent variables, \( X_j(t) = \{X_{ij}(t), \ldots, X_{kj}(t), \ldots, X_{Kj}(t)\} \), associated with ward \( j \) at time period \( t \) in the following well-known specification (Aitkin et al, 1989; Bailey and Gatrell, 1995), with parameters \( \beta(t) = \{\beta_0(t), \beta_1(t), \ldots, \beta_k(t), \ldots, \beta_K(t)\} \)
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estimated by Poisson regression:

\[
\ln D_j(t) = \ln E_j(t) + \beta_0(t) + \sum_k \beta_k(t) X_{kj}(t) + \epsilon_j(t). \tag{3}
\]

The \(E_j\) terms are the expected number of deaths in ward \(j\),

\[
E_j(t) = \sum_{\{as\} \subset AS} r_{as}(t) P_{as}^{as}(t), \tag{4}
\]
given in terms of England and Wales death rates \(r\) at time \(t\) (1981 or 1991) in conjunction with the local population, \(P_{as}^{as}\), partitioned into a set of age—sex categories, \(\{as\} \subset AS\). The natural logarithm of these expected deaths is treated as a constant offset in the estimation process. The \(\epsilon_j\) are Poisson-distributed random errors.

Equation (3) is denoted the free-variable model because the explanatory variables selected and their interrelationship are determined through the estimation process itself. When preselected independent variables are combined in fixed proportions \(w\) to form a deprivation measure \(d\), as, for example, in the Townsend, Carstairs, or Jarman forms (Morris and Carstairs, 1991):

\[
d_j(t) = \sum_k w_k z_{kj}(t), \tag{5}
\]

with the \(z\)-score, defined in terms of the mean \(\mu_k\) and standard deviation \(\sigma_k\) of the distribution of the variable \(X_k\) over the ward system,

\[
z_{kj} = \frac{X_{kj} - \mu_k}{\sigma_k}, \tag{6}
\]

the above equation will be written

\[
\ln D_j(t) = \ln E_j(t) + \beta_0(t) + \beta_1(t) d_j(t) + \epsilon_j(t). \tag{7}
\]

In this composite-variable model the deprivation measure in each ward is given exogenously. The relationship between the estimated deaths in a ward and the Townsend deprivation index \(T_j\) now takes the specific form

\[
\ln \hat{D}_j(t) = \ln E_j(t) + \hat{\beta}_0(t) + \hat{\beta}_1(t) T_j(t), \tag{8}
\]

or

\[
\ln \hat{D}_j(t) = \hat{\beta}_0(t) + \hat{\beta}_1(t) T_j(t), \tag{9}
\]

with \(\hat{\beta}_1(t)\) indicating the gradient of mortality with respect to the deprivation index.

The above models may be estimated separately at each cross section 1981—83 and 1990—92 with the expected deaths calculated at the relevant time period, and with deprivation scores, \(T(81)\) and \(T(91)\), computed with the available census data:

\[
\ln \hat{D}_j(81 - 83) = \ln E_j(81 - 83) + \hat{\beta}_0(81 - 83) + \hat{\beta}_1(81 - 83) T_j(81), \tag{10}
\]

\[
\ln \hat{D}_j(90 - 92) = \ln E_j(90 - 92) + \hat{\beta}_0(90 - 92) + \hat{\beta}_1(90 - 92) T_j(91). \tag{11}
\]

This procedure provides evidence of relative measures of mortality—deprivation gradients separately for each time period. It cannot, however, be adopted to determine the absolute change in death rates between the two time periods, and whether these rates are deprivation dependent, because of the standardisation of the expected deaths and the deprivation index at separate time periods.

2.3 Modelling mortality changes by using Poisson regression

By applying a common standardisation of the expected deaths to one time period (here 1981—83 and indicated by a bar over the expected deaths \(E\)) and exploiting the typically high correlation of \(T(81)\) and \(T(91)\) over the ward system by using a single
independent variable, $T(81)$, a repeated-measures Poisson regression model (Francis et al, 1993) can be used to model mortality changes over time:

$$\ln D_j(t) = \ln \tilde{E}_j(t) + \beta_0 + \beta'_0 \delta(t) + [\beta_1 + \beta'_1 \delta(t)] T_j(81) + \epsilon_j(t),$$

(12)

where $\delta(t)$ is a dummy time period variable taking the value 0 for $t = 1981-83$ and 1 for $t = 1990-92$. In the empirical applications below the extent to which the random error terms are correlated over space and time are examined.

In this repeated-measures design two mortality counts [$D_j(81-83)$, $D_j(90-92)$] are available for each of the $N$ wards. Essentially, this is the model structure used by Staines (1994) and by Shouls et al (1996). Additionally, this repeated-measures model could be represented in a multilevel form (Plewis, 1995), where mortality counts by time period would nest within wards.

The parameter $\beta_0$ is the constant and $\beta_1$ is the gradient of the standardised mortality ratio in 1981–83 with respect to the deprivation index in 1981–83, and their estimated values should be identical to those in the cross-sectional model for 1981–83, equation (10). In the 1990–92 time period, the corresponding constant and gradient are $(\beta_0 + \beta'_0)$ and $(\beta_1 + \beta'_1)$. The parameters $\beta'_0$ and $\beta'_1$ thus represent the change in mortality and the change in the mortality–deprivation gradient (at a deprivation score of zero) between the two time periods. It should be emphasised that the 1990–92 parameters $(\beta_0 + \beta'_0)$ and $(\beta_1 + \beta'_1)$ are not identical to those in equation (11), as equation (12) uses the 1981 Townsend variable and 1990–92 expected deaths standardised to 1981–83.

This repeated-measures model provides independent estimates of deaths in both 1981–83 and 1990–92, and the deprivation indicator in 1981–83 serves as the explanatory variable for the variation of smr at the cross section and for the gradient of its change. By subtracting the estimated number of deaths for $t = 1981-83$ from the corresponding estimate for 1990–92 in equation (12), we can derive the following relationship between the change in smr over time and the deprivation measure

$$\ln \frac{\tilde{D}_j(90 - 92)}{\tilde{D}_j(81 - 83)} = \ln \frac{\tilde{E}_j(90 - 92)}{\tilde{E}_j(81 - 83)} + \hat{\beta}'_0 + \hat{\beta}'_1 T_j(81),$$

(13)

$$\ln \frac{\text{smr}_j(90 - 92)}{\text{smr}_j(81 - 83)} = \hat{\beta}'_0 + \hat{\beta}'_1 T_j(81),$$

(14)

with both mortality ratios standardised to 1981–83.

2.4 Modelling mortality changes by using logit regression

In the repeated-measures Poisson model above, although the estimated deaths summed over all wards equal the observed death totals at the two cross sections 1981–83 and 1990–92 separately, deaths at the ward level are not subject to any a priori conditions which constrain their estimated values. However, alternative forms of the generalised linear model family may be specified according to any additional constraints or conditions which may be imposed. Such conditions may arise for substantive reasons, relating to additional independent information imposing consistency conditions on the estimation of deaths, or be invoked to provide a better statistical fit to the data. In addition to estimating the repeated-measures Poisson models discussed above, we have examined and compared a logit-model representation of mortality changes.

An improved description of the variation in the data is provided by requiring that the sum of the estimated deaths in each ward in 1981–83 and 1990–92 equals the observed deaths:

$$\tilde{D}_j(81 - 83) + \tilde{D}_j(90 - 92) = D_j(81 - 83) + D_j(90 - 92),$$

(15)
for all wards \( j \). The incorporation of this additional set of constraints into the Poisson model (12) may be achieved by replacing the single constant with a set of parameters \( \gamma_j \), one for each ward, and estimating a model of the form

\[
\ln \hat{D}_j(t) = \ln \tilde{E}_j(t) + [\gamma_j + \gamma'_0 \delta(t)] + [\gamma_j + \gamma'_1 \delta(t)] T_j(81) .
\] (16)

Subtraction of the estimated number of deaths for \( t = 1981 \) from the corresponding estimate for 1991 yields the following expression:

\[
\ln \left[ \frac{\hat{D}_j(90 - 92)}{\hat{D}_j(81 - 83)} \right] = \ln \left[ \frac{\tilde{E}_j(90 - 92)}{\tilde{E}_j(81 - 83)} \right] + \gamma'_0 + \gamma'_1 T_j(81) .
\] (17)

It is important to note that, although expression (17) is similar to equation (13), the estimated parameters in the constrained model (17) will be different from those derived from the unconstrained model (13). We thus use \( \gamma \) to denote the parameters in the constrained form to distinguish them from the values of \( \beta \) derived in the unconstrained form.

The incorporation of the constraints (15) into the Poisson model (12) is essentially equivalent to a logit estimation of the change in smr. Following the arguments in Gardner and Altman (1989, pages 60–61) or in Aitkin et al (1989, pages 231–234), as the ward deaths \( D_j(81–83) \) and \( D_j(90–92) \) are considered to be independent random Poisson variables with means \( E_j(81–83) \) and \( E_j(90–92) \), respectively, then the sum \( [D_j(81–83) + D_j(90–92)] \) is also Poisson distributed with mean \( [E_j(81–83) + E_j(90–92)] \). The joint distribution of \( D_j(90–92) \) conditional on the total \( [D_j(81–83) + D_j(90–92)] \), can be considered a binomial variable with sample size \( [D_j(81–83) + D_j(90–92)] \) and proportion \( D_j(90–92)/[D_j(81–83) + D_j(90–92)] \). If we write

\[
P_j(90–92) = \frac{D_j(90–92)}{D_j(81–83) + D_j(90–92)} ,
\] (18)

and

\[
P_j(81–83) = 1 - P_j(90–92) ,
\] (19)

then the ratio of the deaths in the two years may be expressed as an odds ratio

\[
\frac{D_j(90–92)}{D_j(81–83)} = \frac{P_j(90–92)}{1 - P_j(90–92)} ,
\] (20)

with \( P_j \) bound in the interval \([0,1]\). The proportion of deaths, \( P_j \), may now be estimated through logit regression. Adopting a linear specification for the independent variables allows the change in mortality over the decade to be estimated in logit form with \( N – 2 \) degrees of freedom (df).

\[
\ln \left[ \frac{P_j(90–92)}{1 - P_j(90–92)} \right] = \ln \left[ \frac{\tilde{E}_j(90–92)}{\tilde{E}_j(81–83)} \right] + \gamma'_0 + \gamma'_1 T_j(81) + e_j .
\] (21)

Here, the error term is binomially distributed.

For each ward, the sum of the estimated deaths in 1981–83 and 1990–92 derived from expression (21) is consistent with the constraints (15). Thus, if we require constraints (15) to be satisfied, then the change in mortality over the decade may be modelled through the equivalent procedures of logit analysis or a repeated-measures Poisson model incorporating ward-based dummy variables.
2.5 Models with deprivation-class dummy variables

In the above models, the explanatory variables are expressed in continuous form and, in particular, a linear relationship is assumed. This allows a statistical test of the existence of a gradient of mortality at the cross section or a gradient in the temporal change of mortality over the deprivation variable. Nonlinearities may be introduced in various ways, either using continuous variables with locally weighted regression, or with polynomial forms or with splines, or using categorical variables. To provide a formal model for the tabular analyses of Phillimore et al (1994a), which we also used in our first paper (Higgs et al, 1998), we have chosen the categorical approach in which dummy variables are introduced for each quintile. This allows us to assess whether the variation between different deprivation quintile groups is statistically significant, either at the cross section or in relation to temporal changes.

For example, partitioning the continuous deprivation variable, \( d_j \), in the cross-sectional Poisson model (7), into sets \( J_m \) with \( m = 1, \ldots, 5 \) for quintiles and \( 1, \ldots, 10 \) for deciles, then

\[
\ln D_j(t) = \ln E_j(t) + \beta_0(t) + \sum_m \beta_m(t)\delta_j(t) + \epsilon_j(t),
\]

(22)

with

\[
\delta_j = \begin{cases} 1, & \text{if } j \in J_m \\
0, & \text{otherwise}, \end{cases}
\]

can be used to assess whether differential effects exist for the variation of mortality over the quintile or decile groups. Similarly, the dummy variables may be introduced into the repeated measures Poisson and logit models to examine any differential effects of the temporal change in mortality over the deprivation groups. The logit form becomes

\[
\ln \left[ \frac{P_j(90-92)}{1 - P_j(90-92)} \right] = \ln \left[ \frac{E_j(90-92)}{E_j(81-83)} \right] + \gamma_0 + \sum_m \gamma_m \delta_j + \epsilon_j.
\]

(23)

2.6 Further interpretation of the parameters

In Higgs et al (1998), we produced results for the proportional change in the smr over the intercensal period. The above models may be readily manipulated to yield such information. Whether the unconstrained or constrained models are invoked to estimate changes in mortality over time, the estimated variables will be in the form (13). The change in the mortality ratio \( \Delta \text{smr} \) may thus be expressed in terms of the estimated coefficients as follows:

\[
\ln \left[ 1 + \frac{\Delta \text{smr}}{\text{smr}(81-83)} \right] = \hat{\beta}_0 + \hat{\beta}_1 T(81),
\]

(24)

or

\[
\frac{\Delta \text{smr}}{\text{smr}(81-83)} = \exp \left[ \hat{\beta}_0 + \hat{\beta}_1 T(81) \right] - 1,
\]

(25)

with

\[
\Delta \text{smr} = \text{smr}(90-92) - \text{smr}(81-83),
\]

(26)

all mortality ratios being standardised to 1981–83.

The parameter \( \beta_1 \) may be interpreted with reference to the condition \( T(81) = 0 \), which corresponds approximately to the aggregate condition for the whole study area, in this case Wales. So
where the subscript W denotes the national figure. The parameter $\beta'_0$ is thus related to the proportional change in mortality for Wales as a whole, and

$$\hat{\beta}'_0 = \frac{1}{y + 1} \frac{\partial y}{\partial T},$$

with

$$y = \frac{\Delta \text{smr}}{\text{smr}(81 - 83)}.$$

Thus $\beta'_0$ represents the gradient of the proportional decrease in mortality over the deprivation variable.

For those disease classes for which the absolute change in the smr over the decade is small compared with the smr itself then, to a good approximation, we may write the estimated model in linear form

$$\frac{\Delta \text{smr}}{\text{smr}(81 - 83)} \approx \hat{\beta}'_0 + \hat{\beta}' T(81).$$

The parameters $\beta'_0$ and $\beta'_1$ are the intercept and slope of the above straight line with the intercept approximately equal to the proportional national change in smr, standardised to a 1981 base,

$$\hat{\beta}'_0 \approx \frac{\Delta \text{smr}_w}{\text{smr}_w(81 - 83)},$$

and $\beta'_1$, the gradient of the change in smr, over the deprivation variable.

2.7 Summary of modelling strategies

We summarise the modelling strategies as follows:

(a) For the estimation of gradients in mortality at the cross sections, Poisson models (3) and (7) are used. Nonlinear dependency of mortality on deprivation classes can be explored by using the dummy variable model (22).

(b) To estimate the changes in mortality over time and gradients in this relationship with respect to deprivation, the unconstrained repeated-measures Poisson model (12) and its constrained version (16) are both adopted. The latter is equivalent to the logit model (21), which has the form of equation (23) if nonlinear effects of deprivation on mortality are to be examined.

The above model specifications are used to establish statistical relationships between mortality by all causes and standard deprivation measures. Extensions to consider disaggregation by International Classification of Disease (ICD) group is straightforward and involves cause-specific parameters and mortality variables.

3 Application of cross-sectional models

3.1 Ward-level mortality variations in Wales

In a region of predominantly low population density such as Wales a considerable proportion of the wards are characterised by a low death count, even after the three-year aggregates around the census years have been taken. Indeed, many are less than the threshold of 20 deaths below which the mortality ratios are subject to significant variability (OPCS, 1993). Figure 1 shows the 1981-83 distributions of (all-cause) SMR for the age group 0-64, for the number of deaths greater than and less than (or equal to) 20. These provide similar information to that found in the analysis of Thomas and...
High levels of premature mortality are observed in
(a) industrial South and North Wales, with particular concentrations in the valleys of
South Wales and in Northeast Wales;
(b) the small rural towns of North and Mid Wales such as Blaenau Ffestiniog, Dolgellau,
Lampeter, Llandrindod Wells, Caernarfon, Bangor, Fishguard, and Carmarthen, and
the resort towns of North Wales, such as Llandudno, Colwyn Bay, and Rhyl.
(c) the poorer inner-city areas and suburbs of Cardiff, Swansea, Newport, and Wrexham.
A low incidence of premature mortality in 1981 is found in
(d) the rural areas of Wales (although as we note above many of these have fewer than
20 deaths);
(e) the affluent parts of the major towns and cities.
These findings are consistent with the findings of Howe (1986) at the district level and
of Richards (1987) which demonstrate the high death rates in substantial parts of
industrial South Wales. In broad terms, the patterns for the age group 0–64 in

Figure 1. Standardised mortality ratio (SMR) 1981–83 (all causes, 0–64 years): (a) more than 20
deaths, (b) 20 deaths or fewer.

Kaul (1988) and Thomas (1990), with differences attributable to the treatment of
institutional deaths (Thomas and Kaul including this component).
Figure 1 (continued)

1990–92 [not shown here (see Williams et al, 1997)] have a form similar to those of the early 1980s.

3.2 Models relating mortality and deprivation

To quantify the association between premature (0–64) mortality and deprivation and assess the significance of a range of variables at the cross sections, we use the Poisson regression models in composite [equation (7)], dummy [equation (22)], and free-variable [equation (3)] forms.

3.2.1 Dummy and composite deprivation-variable models

Inclusion of the 1981 Townsend index of material deprivation by using dummy quintiles as the sole explanatory variable produces the following parameter estimates for model (22)

\[
\ln \text{smr}_{(81-83)} = -0.1341 + 0.1002Q_4 + 0.1969Q_3 + 0.2587Q_2 + 0.3410Q_1 \quad (31)
\]

with standard errors given in parentheses. \(Q_1\) refers to the most deprived quintile of wards, and so on. All parameter estimates are significant. The exponential of the reference parameter, \(-0.1341\), provides an estimate of 0.8745 for the smr of the least deprived (fifth) quintile. The smrs for the other quintiles are obtained by taking the
exponential of the sum of the appropriate quintile parameter and the above reference parameter. When multiplied by 100, these smr estimates are nearly identical to the rounded SMRs given in our first paper [see table I(a) in Higgs et al, 1998]. These parameter estimates suggest a roughly linear increase of smr over the ordered deprivation quintiles. The flexibility of a statistical approach means that a continuous, linear deprivation effect can easily be estimated from

\[
\ln \text{smr}_j(81 - 83) = 0.0442T_j(81) .
\]

Although the constant term \( \beta_0 \) is not significant at the 5% level (and has therefore been constrained to a zero value in a reestimated model), the deprivation variable is highly significant. The scaled deviance (Aitkin et al, 1989) measure of unexplained variation in ward mortality is 982.7 at 799 df compared with 1320.8 (also with 799 df) for a constant-only (null) model. This is an improvement on the dummy-variable model (31) whose scaled deviance measure is 997.0 at 795 df. However, neither model is a particularly good fit as the scaled deviances are not approximately equal to the degrees of freedom. However, this is not surprising as they incorporate only one explanatory variable. Goodness of fit can be reassessed with the free-variable models below.

To test the sensitivity of model (32) to variation in the deprivation measure, the Townsend index has been replaced by the 1981 Carstairs and Jarman measures and their results compared. In terms of the scaled deviance values, these two alternative deprivation indicators are significant, but no better than the Townsend measure, in explaining the variation of premature mortality over the ward system in Wales.

For 1990–92 the corresponding models relating the mortality ratio, standardised to 1991, to the deprivation index, calculated with 1991 Census data reconstituted to 1981 ward boundaries (Atkins et al, 1993), are given by

\[
\ln \text{smr}_j(90 - 92) = -0.2215 + 0.0818Q_4 + 0.1637Q_3 + 0.2685Q_2 + 0.4070Q_1 .
\]

\[
(0.0211) (0.0288) (0.0272) (0.0264) (0.0248)
\]

\[
\ln \text{smr}_j(90 - 92) = -0.0540 + 0.0546T_j(91) .
\]

\[
(0.00811) (0.00262)
\]

There is a significant mortality—deprivation gradient at the 1990–92 cross section. The scaled deviance statistics are 1176 (at 795 df) for the dummy-quintile model (33) and 1135 (at 798 df) for model (34) compared with 1562 (at 799 df) for the null model. Once again the linear deprivation model (34) is an improvement on the dummy-quintile representation.

The relations (32) and (34) are graphically displayed in figure 2. It is difficult to compare these two equations in the light of the standardisation of mortality and deprivation to different years. What can be said is that the gradient of the mortality ratio with respect to the corresponding deprivation index is greater in 1991 than in 1981.

3.2.2 Free-variable models

In the above models the number of explanatory variables is, by construction, reduced to one, the deprivation score. In order to assess the differential significance of the constituent variables of the deprivation measure, or any other factors, in explaining the spatial variation of the mortality ratio, we now consider the free-variable model. In addition to the range of variables from which the Townsend measure is determined, two 'mining variables' are considered to assess the legacy of the coalmining industry in Wales. These are the percentage of male employed residents in nonoffice jobs in the coal extraction and solid fuel manufacturing industries in 1981, and the percentage of employed residents in the mining and quarrying industries in 1971.
Interestingly, the specification which provided the best statistical fit to the variation in SMR in 1981–83 is given by

\[ \ln \text{SMR}_{(81-83)} = -0.317 + 0.00732(\%\text{NOCAR}) + 0.00823(\%\text{NOJOB}) \]

The 'absence of a job' (\%\text{NOJOB}) and 'absence of a car' (\%\text{NOCAR}) in a household are the only significant variables. The non-owner-occupier, overcrowding, and 'mining' variables were not statistically significant at the ward level for the all-causes 0–64 mortality variable. Although the scaled deviance measure of 929 (at 797 df) indicates that this is an improvement over model (32) with the composite Townsend variable, this is still not a well-fitting model. Other explanatory variables need to be considered.

The corresponding best-fitting relation for 1990–92 is very similar as the overcrowding variable (\%\text{OVERCROWDING}) is only marginally significant:

\[ \ln \text{SMR}_{(90-92)} = -0.415 + 0.00905(\%\text{NOCAR}) + 0.00779(\%\text{NOJOB}) + 0.0241(\%\text{OVERCROWDING}) \]

All the explanatory variables refer to their 1991 Census values. The scaled deviance measure is 1111 at 796 df, suggesting only a modest improvement over the composite variable model (34).
3.3 Summary and discussion

Material deprivation, whether it is measured by the Townsend, Carstairs, or Jarman indicator, is highly significant in explaining the spatial variation of premature mortality in Wales. However, all the models discussed are characterised by a large degree of unexplained variation (as measured by the scaled deviance function) and deprivation, by itself, does not result in a good explanation of the dispersion. The value of the Spearman rank correlation coefficient for the 1990–92 0–64-year smr and the 1991 Townsend measure is only 0.53 [although, as noted by Phillimore et al (1994b) the grouping of mortality data over a wider span of years can markedly improve this measure].

In particular, many of those wards which are responsible for the largest (standardised) residuals in the models are rural in nature and are often associated with a small number of deaths. Their removal substantially increases the Spearman correlation statistic. The value for the four most urbanised counties in South Wales in 1990–92 is 0.65. For the Bro Taf Health Authority, which contains some of the most marked contrasts in both mortality and deprivation in Wales, the correlation between the all-age smr for 1988–94 and the Townsend index is 0.71.

The large unexplained residual variation in the all-Wales models suggests the possible absence of important explanatory variables, the presence of distributional effects within the wards themselves which vary between low-density and high-density wards, or deviation from the assumptions underpinning the Poisson model. These features are not unexpected in such aggregate statistical models.

4 Mortality changes: applying the repeated-measures Poisson model

This is the first of three sections in which we apply models which explore the variation of mortality over the wards of Wales in the two time periods, 1981–83 and 1990–92. To achieve comparability over time, expected deaths for both the 1981–83 and 1990–92 time periods are computed with 1981–83 death rates for England and Wales. Following Phillimore et al (1994a; 1994b) we relate mortality change only to deprivation in 1981, given the high correlation (0.88 for the 800 Welsh wards) between the 1981 and 1991 Townsend scores. The data are identical to those used in Higgs et al (1998).

In this section we present results from using the repeated-measures Poisson model [equation (12)]. In table 1 (over) the parameter estimates for this Poisson model are given for various causes, age, and gender classes used in Higgs et al (1998). It should be recalled that the parameters $\beta_0$ and $\beta_1$ are the constant and gradient at the 1981–83 cross section, respectively, and $\beta'_0$ and $\beta'_1$ represent the change in mortality and change in the mortality–deprivation gradient (at the national average deprivation score of zero) over the decade. Because of the log-link function in Poisson regression models these parameters relate to the natural logarithm of smrs. The exponential of these parameter values (see table 1) can be interpreted directly in terms of smrs. Thus $\exp \beta_0$ provides an estimate of the 1981–83 smr at a zero Townsend deprivation score; $\exp \beta_1$ represents the multiplier on the 1981–83 smr for a unit increase in the deprivation score; $\exp \beta'_0$ is the multiplier for the change in smr between 1981–83 and 1990–92; and $\exp \beta'_1$ is the multiplier for the change in smr caused by the changing influence from 1981–83 to 1990–92 of a unit difference in the 1981 deprivation score.

The $\beta_1$ estimates in table 1(a) reveal that, for all age–sex–cause subsets, the Townsend deprivation variable has a significant association with mortality in 1981–83. The signs on the parameters indicate, as expected, worsening mortality with higher deprivation, except for breast cancer where there is a well-known reverse gradient with social class. The $\beta'_0$ values in table 1(b) suggest significant declines in mortality from 1981–83 to 1990–92, with the sole exception of males aged 15–44, for whom there is no significant change
in all-cause mortality (compare Phillimore et al, 1994a), and a significant increase in suicides. The positive \( \beta_1 \) estimates imply that the mortality–deprivation gradient has steepened over the decade for all-cause mortality, and for deaths due to circulatory diseases, of those aged 0–64 and 0–74. For deaths from cancers and suicide the mortality–deprivation gradient has not changed significantly between the two time periods.

These statistical relationships can be displayed graphically (for example, figure 3), by plotting the natural logarithm of smr against the Townsend deprivation score. Two lines record the relationships between mortality and deprivation at the two cross sections. Declining (increasing) mortality over the decade results in the fitted linear relationship for 1990–92 being below (above) that for 1981–83. The separation of the lines is a measure of the change in mortality at a particular deprivation level. Where the mortality gradient has increased (as in figure 3) the steeper slope of the line for 1990–92 indicates widening mortality differentials over the decade, with greater mortality decreases in affluent than deprived wards. Where there has been no significant change in the mortality–deprivation gradient, the 1981–83 and 1990–92 lines are parallel as for deaths from cancer and from suicide (graphs not shown). For males aged 15–44, the all cause mortality–deprivation line (graph not shown) is the same in 1981–83 and 1990–92, indicating change in neither the level of mortality nor in its relationship with deprivation.

The graphs display substantial residual variation about the estimated relationship. As for the cross-sectional models, an examination of the standardised residuals suggests rural wards with low numbers of deaths contribute disproportionately to this total residual variation. Improved specifications to the models, including urban and rural contextual effects, are currently under investigation in another study.

Figure 3. Standardised mortality ratio (SMR) from the repeated-measures Poisson model (all causes, 0–64 years).
Table 1. Parameter estimates for the repeated-measures Poisson model [equation (12) in the text].

| Cause and age group          | $x_0$   | SE    | $\exp x_0$ | 95% CI lower | 95% CI upper |
|-----------------------------|---------|-------|-------------|--------------|--------------|
| (a) $x_0 = \beta_0$; $x_1 = \beta_1$ |         |       |             |              |              |
| All causes, 0–64            | 0.0133 ns | 0.00762 | 1.013 | 0.998 | 1.029 |
| All causes, 0–74            | 0.0273 | 0.00509 | 1.028 | 1.017 | 1.038 |
| All causes, male, 15–44     | -0.0791 | 0.0185 | 0.924 | 0.891 | 0.958 |
| All causes, female, 65–74   | 0.0320 | 0.0108 | 1.032 | 1.011 | 1.055 |
| Cancers, 0–64               | -0.00981 ns | 0.0126 | 0.990 | 0.966 | 1.015 |
| Cancers, 0–74               | -0.0273 | 0.00884 | 0.973 | 0.956 | 0.990 |
| Breast cancer, 0–64         | 0.105 | 0.0329 | 1.110 | 1.041 | 1.184 |
| Breast cancer, 0–74         | 0.0646 | 0.0267 | 1.067 | 1.012 | 1.124 |
| Circulatory, 0–64           | 0.0538 | 0.0121 | 1.055 | 1.030 | 1.081 |
| Circulatory, 0–74           | 0.0773 | 0.00742 | 1.080 | 1.065 | 1.096 |
| Suicides, male, 15–44       | -0.0612 ns | 0.0748 | 0.941 | 0.812 | 1.089 |
| Suicides and injury         | -0.1667 | 0.0664 | 0.846 | 0.743 | 0.964 |
| undetermined, male, 15–44   |         |       |             |              |              |

(b) $x_0 = \beta'_0$; $x_1 = \beta'_1$ |         |       |             |              |              |
| All causes, 0–64            | -0.280 | 0.0115 | 0.756 | 0.739 | 0.773 |
| All causes, 0–74            | -0.236 | 0.0075 | 0.789 | 0.778 | 0.801 |
| All causes, male, 15–44     | ns      |       |             |              |              |
| All causes, female, 65–74   | -0.177 | 0.0158 | 0.838 | 0.812 | 0.864 |
| Cancers, 0–64               | -0.126 | 0.0170 | 0.881 | 0.852 | 0.911 |
| Cancers, 0–74               | -0.037 | 0.0116 | 0.964 | 0.942 | 0.986 |
| Breast cancer, 0–64         | -0.206 | 0.0466 | 0.814 | 0.743 | 0.892 |
| Breast cancer, 0–74         | -0.119 | 0.0368 | 0.888 | 0.826 | 0.954 |
| Circulatory, 0–64           | -0.452 | 0.0192 | 0.637 | 0.613 | 0.661 |
| Circulatory, 0–74           | -0.367 | 0.0113 | 0.693 | 0.678 | 0.709 |
| Suicides, male, 15–44       | 0.3241 | 0.0901 | 1.383 | 1.159 | 1.650 |
| Suicides and injury         | 0.4668 | 0.0777 | 1.595 | 1.370 | 1.857 |
| undetermined, male, 15–44   |         |       |             |              |              |

Note: CI confidence interval; ns indicates that a parameter is not significantly different from zero; SE standard error.

In the above analysis it was assumed that the random residuals are independently distributed over space and time. The spatial structures of the residuals at the two cross sections were mapped to check for spatial autocorrelation, but this was not found to be a serious problem. Additionally, correlating 1981–83 with 1990–92 standardised residuals revealed only weak associations: for example a correlation coefficient of 0.18 obtained with residuals from the analysis of all-cause, 0–64 mortality.

5 Mortality changes: applying the logit model

The binomial logit model [equation (21)] has parameters, $\gamma'_0$ and $\gamma'_1$, representing the change in the standardised mortality ratio and in its gradient with respect to deprivation. Comparing tables 1 and 2 reveals very little difference (in this empirical illustration) between the estimated values of the parameters ($\beta'_0$, $\beta'_1$) and ($\gamma'_0$, $\gamma'_1$). This indicates that the presence of the constraints (15) produces little difference between the results of the repeated-measures Poisson model without constraints [equation (12)] and the binomial logit model [equation (21)]. Either form may be used to estimate the change in mortality and in its gradient with respect to deprivation.
Table 1 (continued)

| $x_i$ | SE   | $\exp x_i$ | 95% CI lower | 95% CI upper |
|-------|------|------------|--------------|--------------|
| 0.042 | 0.00227 | 1.043 | 1.038 | 1.048 |
| 0.0372 | 0.00157 | 1.038 | 1.035 | 1.041 |
| 0.0452 | 0.00557 | 1.046 | 1.035 | 1.058 |
| 0.0303 | 0.00344 | 1.031 | 1.024 | 1.038 |
| 0.0300 | 0.00300 | 1.030 | 1.024 | 1.036 |
| 0.0305 | 0.00207 | 1.031 | 1.027 | 1.035 |
| -0.0195 | 0.00829 | 0.981 | 0.965 | 0.997 |
| -0.0150 | 0.00663 | 0.985 | 0.972 | 0.998 |
| 0.0500 | 0.00359 | 1.051 | 1.044 | 1.059 |
| 0.0380 | 0.00229 | 1.039 | 1.034 | 1.043 |
| 0.0526 | 0.01554 | 1.054 | 1.022 | 1.087 |
| 0.0616 | 0.01316 | 1.064 | 1.036 | 1.091 |
| 0.00951 | 0.00344 | 1.0096 | 1.0028 | 1.0164 |
| 0.0118 | 0.00231 | 1.0119 | 1.0073 | 1.0165 |
| ns | 0.00497 | 1.0194 | 1.0096 | 1.0294 |
| ns | 0.00569 | 1.0173 | 1.0061 | 1.0287 |
| ns | 0.00349 | 1.0166 | 1.0097 | 1.0236 |

Figure 4 (over) is the graphical equivalent for the logit model of figure 3 for the repeated-measures Poisson model. Because of the logit link function, the vertical axis of the graph measures the natural logarithm of the odds ratio of estimated smr in 1990–92 to that in 1981–83. A single line provides an estimate of the change in mortality as a function of deprivation. Its position below the horizontal axis indicates the extent of the fall in mortality between 1981–83 and 1990–92. Its slope reflects changes in the mortality–deprivation gradient over the decade. Thus, a zero slope (flat) line, as for deaths from cancers and from suicide (not shown), indicates no such change, whereas a positive slope, as in figure 4, reveals widening mortality differentials with respect to deprivation.

The parameter estimates for $\gamma_0$ in table 2 reflect the change in mortality between 1981–83 and 1990–92 at the Townsend deprivation score of zero. The proportional change in mortality is given directly by the odds ratio minus one. Mortality decline is evident for all disease categories, with the exception of deaths of males aged 15–44 from all causes (no significant change) and from suicides (significant increase). Because the mean Townsend score for Wales as a whole is close to zero, these estimated declines in mortality should be (and are) very similar to those for the whole of Wales calculated in the tabular analyses of our first paper (Higgs et al, 1998).
Table 2. Parameter estimates for the logit model [equation (21) in the text].

| Cause and age group          | $\gamma_0$ | SE       | Odds ratio, $\exp(\gamma_0)$ | 95% CI   | Odds ratio $-1$ |
|------------------------------|------------|---------|-------------------------------|---------|-----------------|
| All causes, 0–64             | -0.2735    | 0.01154 | 0.7607                        | 0.7437  | 0.7781          |
| All causes, 0–74             | -0.2312    | 0.00756 | 0.7936                        | 0.7819  | 0.8054          |
| All causes, male, 15–44      | -0.05142   | 0.03195 | 0.950                         | 0.892   | 1.011           |
| All causes, female, 65–74    | -0.1783    | 0.0159  | 0.8367                        | 0.8109  | 0.8633          |
| Cancers, 0–64                | -0.1233    | 0.0171  | 0.8840                        | 0.8549  | 0.9141          |
| Cancers, 0–74                | -0.03418   | 0.0117  | 0.9664                        | 0.9445  | 0.9888          |
| Breast cancer, 0–64          | -0.2032    | 0.0467  | 0.8161                        | 0.7447  | 0.8943          |
| Breast cancer, 0–74          | -0.1156    | 0.0369  | 0.8908                        | 0.8287  | 0.9576          |
| Circulatory, 0–64            | -0.4435    | 0.0194  | 0.6418                        | 0.6179  | 0.6666          |
| Circulatory, 0–74            | -0.3622    | 0.0114  | 0.6961                        | 0.6807  | 0.7119          |
| Suicides, male, 15–44        | 0.3179     | 0.0910  | 1.374                         | 1.1497  | 1.6426          |
| Suicides and injury          | 0.4649     | 0.0782  | 1.592                         | 1.3656  | 1.8555          |
| undetermined, male, 15–44    | ns         | 0.0167  | ns                            | 0.0167  | ns              |

Note: CI confidence interval; ns indicates that a parameter estimate is not significantly different from zero; thus the odds ratio is not significantly different from 1 and the odds ratio $-1$ is not significantly different from zero; SE standard error.

The estimated $\gamma_0$ values measure the effect of a unit change in the 1981 deprivation score on changing mortality. Expressed as odds ratios, they represent multipliers. Thus for deaths from circulatory diseases, each unit change of deprivation changes the odds ratio calculated at any given deprivation score by 1.68% (for deaths in the age range 0–64) and by 1.70% (for those in the range 0–74). The impact of deprivation is significant only for all-cause changes in mortality (excepting males aged 15–44) and for circulatory diseases. The positive signs on the parameter estimates signify that the mortality–deprivation relationship is steeper in 1990–92 than in 1981–83. In turn, this means that mortality differentials have widened, with the most deprived wards experiencing the smallest declines in mortality and the least deprived the largest reductions.

Figure 4. Change in all-causes (0–64 years) standardised mortality ratio (SMR) from the logit model, 1981–83 to 1990–92.
Table 2 (continued)

| $\gamma_i$ | SE       | Odds ratio, exp $\gamma_i$ | 95% CI       |
|------------|----------|-----------------------------|--------------|
| 0.00876    | 0.003489 | 1.0088                      | 1.0019 - 1.0157 |
| 0.01118    | 0.00235  | 1.0119                      | 1.0072 - 1.0165 |
| ns         |          |                             |              |
| 0.02049    | 0.005092 | 1.0207                      | 1.0106 - 1.0309 |
| ns         |          |                             |              |
| ns         |          |                             |              |
| ns         |          |                             |              |
| 0.0167     | 0.005791 | 1.0168                      | 1.0054 - 1.0284 |
| 0.01681    | 0.003556 | 1.0170                      | 1.0099 - 1.0241 |
| ns         |          |                             |              |
| ns         |          |                             |              |

6 Mortality changes: applying the logit model with dummy deprivation categories

The models applied in sections 4 and 5 assume a continuous linear form for the deprivation effect. As for the cross-sectional models in section 3, possible nonlinear deprivation influences can be examined by using dummy variables representing wards categorised on their deprivation scores. Choosing ward quintiles as these categories produces a formal statistical representation of the tabulations of mortality change by deprivation classes used in our first paper (Higgs et al, 1998).

Table 3 (over) gives the result of applying the logit model with deprivation class dummies [equation (23)]. Its purpose is to identify those groups of deprivation quintiles which are significantly different from each other. Thus the result \{3,4,5\} \{1,2\} for deaths due to circulatory diseases of those aged 0–74 indicates that there is no significant difference in the mortality change between 1981–83 and 1990–92 of quintiles 1 and 2 on the one hand, and, on the other, of quintiles 3,4, and 5 (1 = most deprived quintile of wards, 5 = least deprived quintile of wards). However, there is a significant difference between these two sets of quintiles, with residents in the two most deprived quintiles of wards having a significantly smaller change in mortality over the decade than those in the other three quintiles.

The first group in each application presented in table 3 is the reference group of quintiles. The odds ratio minus one is the model's estimate of the proportional change in mortality over the decade for the reference group. For example, deaths from all causes for those aged 0–74 have declined for the two least deprived quintiles (4 and 5) by 22.6%. Parameters for the second and third groups (where relevant) measure differences in changing mortality from that of the reference group. Hence, to find the estimate of mortality change for a nonreference group its parameter must be added to the parameter of the reference group before calculating the odds ratio minus one. Again if we use premature (0–74) deaths from all causes, residents in deprivation quintiles 2 and 3 experienced an 18.9% reduction in mortality over the decade, whereas for those in the most deprived quintile (1) the decline in mortality was 16.2%.
Table 3. Parameter estimates for the logit models with deprivation-quintile dummy variables [equation (23) in the text].

| Cause and age group | Groupa | 1st | 2nd | 3rd | 1st parameter | odds ratio | odds ratio —1 |
|---------------------|--------|-----|-----|-----|----------------|------------|--------------|
| All causes, 0 – 64  | 2,3,4,5| 1   |     |     | —0.282         | 0.754      | —0.246       |
|                     | 4,5    | 2,3 | 1   |     | —0.2565        | 0.774      | —0.226       |
| All causes, 0 – 74  | 1,2,3,4,5| 1,2,3 | 1   |     | —0.05142       | 0.950      | —0.050       |
| All causes, male, 15 – 44 | 4,5    | 1,2,3 |     |     | —0.2307        | 0.794      | —0.206       |
| All causes, female, 65 – 74 | 4,5    | 1,2,3 |     |     | —0.1233        | 0.884      | —0.116       |
| Cancers, 0 – 64    | 1,2,3,4,5| 1,2,3,4 |     |     | —0.111         | 0.895      | —0.105       |
| Cancers, 0 – 74    | 5      | 1,2,3,4 |     |     | —0.2032        | 0.816      | —0.184       |
| Breast cancer, 0 – 64 | 1,2,3,4,5|     |     |     | —0.212         | 0.809      | —0.191       |
| Breast cancer, 0 – 74 | 1,5    | 2,3,4 |     |     | —0.454         | 0.635      | —0.365       |
| Circulatory, 0 – 64 | 2,3,4,5| 1    |     |     | —0.383         | 0.682      | —0.318       |
| Circulatory, 0 – 74 | 3,4,5  | 1,2  |     |     | —0.309         | 0.619      | —0.275       |
| Suicides, male, 15 – 44 | 1,2,3,4,5|     |     |     | —0.3179        | 1.374      | 0.374        |
| Suicides and injury undetermined, male, 15 – 44 | 1,2,3,5| 4    |     |     | 0.5317         | 1.702      | 0.702        |

Note: ns indicates that a parameter estimate is not significantly different from zero; thus the odds ratio is not significantly different from 1 and the odds ratio —1 is not significantly different from zero. Standard errors are given in parentheses.

For none of the disease classes used are significant differences in changing mortality found between all five quintiles, unlike the findings for the cross-sectional models reported in section 3. At most, three groups are needed, but only in one example to describe all-cause mortality changes in the age range 0 – 74. Indeed, there are no differences between any of the quintiles for deaths from all causes and from suicides (ICD E950 – E959 only) involving males aged 15 – 44, and for premature (0 – 64) deaths from cancer and from breast cancer. Typically the five quintiles can be grouped into just two sets with significantly different changes in mortality.

Comparing the findings from the models with linear deprivation effects (table 2) with those in table 3, reveals the following.

1) For premature (0 – 64) deaths from all causes and from the subset of circulatory diseases, the most deprived quintile stands out as experiencing significantly lower declines in mortality than the other four quintiles. The significant linear influence of deprivation in the table 2 results is shown to be concealing the finding in table 3 of no significant deprivation influence on mortality change across those four quintiles.

2) Although table 2 reveals no significant linear effect of deprivation on changing mortality from all cancers, from breast cancer, and from suicides, table 3 brings out
Table 3 (continued)

| 2nd group | 3rd group |
|-----------|-----------|
| 2nd parameter | sum of 2nd and 1st parameters | odds ratio | odds ratio |
| 0.069 (0.0209) | -0.213 (0.0170) | 0.808 | -0.192 |
| 0.0475 (0.0161) | -0.209 (0.0104) | 0.811 | -0.189 |
| 0.117 (0.0308) | -0.1137 (0.0164) | 0.893 | -0.107 |
| 0.0892 (0.0337) | -0.0218 (0.0126) ns | 0.978 | -0.022 ns |
| 0.166 (0.0747) | -0.046 (0.0484) ns | 0.955 | -0.045 ns |
| 0.116 (0.0346) | -0.338 (0.0279) | 0.713 | -0.287 |
| 0.0893 (0.0200) | -0.2937 (0.0137) | 0.746 | -0.255 |
| -0.4649 (0.2205) | 0.0668 (0.2036) ns | 1.069 | 0.069 |

| 3rd parameter | sum of 3rd and 1st parameters | odds ratio | odds ratio |
| 0.0795 (0.0169) | -0.177 (0.0116) | 0.838 | -0.162 |

a The numbers in the column indicate: 1 the most deprived quintile of wards; 5 the least deprived quintile of wards.

a nonlinear influence for 0–74 deaths from all cancers and from breast cancer and for suicide and indeterminate deaths among males aged 15–44.

Thus, although a linear, rather than a dummy variable, representation of the influence of deprivation on mortality appears preferable for the cross-sectional models (section 3), nonlinear specifications are essential for models of mortality change.

7 Conclusions

Employing ward data we have used a generalised linear model as a unifying framework for exploring the relationship between mortality and deprivation both at the cross section and over time. This allows a formal representation of the tabular analyses used by Phillimore et al (1994a), but also permits more flexible and varied analyses to be undertaken. Thus the significance of a wider variety of variables and of nonlinear arrangements of categories can be assessed. The tabular analysis undertaken in our first paper (Higgs et al, 1998) can therefore be seen as a special member of the family of statistical models considered in this paper. We have used the repeated-measures Poisson model and have introduced the binomial logit model and have found them to be particularly flexible and transparent methods for exploring mortality change, which is far from uniform over disease classes. Furthermore, the exploration of nonlinear mortality – deprivation relationships is
particularly important in discerning specific area deprivation groups which are lagging behind in the general trend of declining premature mortality.

Through the provision of a statistical representation for tabular analyses of mortality and deprivation, a wider range of statistical models can now be accessed. In particular, an important extension for our future research is the application of random effects models (Congdon, 1995; Goldstein, 1995; Longford 1993) to include a representation of possible contextual effects, such as urban—rural differences in mortality.

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