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Bayesian estimation of long-term health consequences for obese and normal-weight elderly people

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Summary. Obesity is a rapidly growing public health problem even among the elderly. Understanding the disabling consequences of obesity in the elderly will help us to design better effective intervention management guidelines for the elderly obese. To examine the long-term health consequences of the obese elderly, we present a joint model consisting of two bivariate ordered responses observed at successive time points. The bivariate ordered response model corresponds to the subject's self-reporting health status outcomes including self-rated health and functional status. Although the joint model that we propose is generally suited for use in health and disease research, where the ordered value responses are observed at successive time points, we further extend it by addressing some of the challenges by incorporating the semiparametric features in the ordinal logistic model, by modelling the underlying latent states of health that are associated with self-rated health, by jointly modelling the bivariate ordinal outcomes to mitigate the variability of the single response and by accounting for the non-ignorable missing data due to different reasons through a multinomial logit model. The motivating data were obtained from the Second Longitudinal Study of Aging, which are longitudinal survey data from 1994–2000 providing various useful information on the health status of elderly people. Parameter estimation of our joint model was performed in a Bayesian framework via Markov chain Monte Carlo methods. Analytical results demonstrate the difference in longitudinal patterns of the health outcomes between the two weight groups, validating our hypothesis that different management strategies for the obese elderly should be employed.

Keywords: Bayesian estimation; Markov chain Monte Carlo methods; Missing data; Obesity; Ordered logistic regression; Ordinal response; Splines

1. Introduction

The prevalence of obesity is one of the fastest growing epidemics in the USA and the rest of the world. As the life expectancy in the USA has been on the rise for a decade, so has the prevalence of obesity in the elderly risen as well. Whereas the rate of obesity of the baby boomer generation was 11–16% in the early 1960s, in 2010, no state in the USA had a rate of obesity less than 20%.

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This may be due to the negative effects of current technology-driven lifestyles, the dominance of consumption of food high in fat and carbohydrates, and a general decrease in physical activity.

Incidentally, obesity is not only a growing social problem, but also an important health problem. Obesity is a well-known risk factor for several chronic diseases, including diabetes, hypertension, high cholesterol, stroke, heart disease, certain cancers and arthritis. A recent study (Cawley and Meyerhoefer, 2012) suggests that obesity accounts for nearly 21% of the total US healthcare spending. In light of these facts, there has been a growing interest in the scientific community to understand better how obesity and other contributing factors affect the health of elderly people. Proper evaluation and modelling of the health status of obese elderly people could not only alert them to their own risks, but also prevent their health from growing worse.

Although many researchers have already studied the development of health status late in life (Hong and He, 2010; Lafortune *et al.*, 2009), few have investigated aging with a long-term health status among the obese population. Two important indicators of health status in old age are functional status (FS) and self-rated health (SRH) (Hoeymans *et al.*, 1997). If FS diminishes, one can no longer perform household or self-care activities independently. SRH describes how a person perceives his or her own health and is an indicator of wellbeing or quality of life (Hoeymans *et al.*, 1997). Furthermore, both FS and SRH are important predictors of mortality.

A few previous researchers have identified a relationship between the level of FS and SRH for older adults (Liang *et al.*, 2007; Shooshtari *et al.*, 2007). Among those who proposed joint models for FS and SRH, Hubbard *et al.* (2009) described the longitudinal relationship between these factors. However, the joint relationship between FS and SRH was considered only for general older adults, not for the obese elderly. In addition, in research into aging some complications such as non-ignorable dropouts due to competing risks and possible non-linear effects of predictor variables do not arise from a distinct or separate setting. To understand the whole dynamics of health outcomes better, it is necessary to develop a unified statistical model. In this paper, we aim to bridge this gap in understanding the longitudinal health outcomes of the obese elderly and explore the gradual change in health status of the elderly in the long term by jointly modelling FS and SRH.

Our motivating data set comes from the Second Longitudinal Study on Aging, LSOA II. All respondents were at least 70 years of age at the time of the interview in 1994. The same subjects had follow-up interviews in 1997–1998 and 1999–2000.

The data and the programs that were used to analyse them can be obtained from

`http://wileyonlinelibrary.com/journal/rss-datasets`

Although our main focus lies with obese elderly subjects, we also present the analysis on normal weight subjects to compare the relative influence of factors with the health outcome between the two groups. The sample characteristics of obese and normal weight subjects as shown in Table 1 indicates that there are unequal proportions in race, sex and the level of education; there are demographic and social disparities between these two groups; health status is generally poor and more heterogeneous in the obese group, implying that the separate analysis would be more beneficial.

1.1. Analytical issues of longitudinally measured ordinal health status of the elderly

In epidemiological studies of aging, measurement of multiple outcomes is often recorded over time. Thus, it is important to develop a longitudinal model for these outcomes. However, various issues that have arisen from research into aging complicate longitudinal ordinal response modelling. We summarize below the key challenges and propose our approach to fix the issues.

Table 1. Proportions of non-missing obese and normal weight subjects of the LSOA II study for each characteristic†

Variable	*Results for obese group*			*Results for normal weight group*		
	Wave 1	*Wave 2*	*Wave 3*	*Wave 1*	*Wave 2*	*Wave 3*
White	0.82	—	—	0.92	—	—
Female	0.69	—	—	0.61	—	—
Less than high school education	0.32	—	—	0.22	—	—
Age	76.09	—	—	78.21	—	—
Fewer than 3 conditions	0.62	0.55	0.37	0.74	0.75	0.61
FS						
(a) independent	0.48	0.34	0.28	0.62	0.52	0.48
(b) IADL disabled only	0.11	0.11	0.09	0.11	0.13	0.12
(c) moderately ADL disabled	0.22	0.34	0.35	0.14	0.22	0.21
(d) severely ADL disabled	0.19	0.21	0.28	0.13	0.13	0.18
SRH						
(a) excellent	0.08	0.07	0.06	0.15	0.14	0.11
(b) very good	0.20	0.21	0.17	0.26	0.29	0.29
(c) good	0.36	0.32	0.34	0.34	0.33	0.33
(d) fair	0.25	0.28	0.28	0.17	0.17	0.19
(e) poor	0.10	0.12	0.15	0.08	0.07	0.08

†The *t*-test showed a statistically significant difference between the proportions of the obese and normal weight group for demographic and social variables such as race, sex and education.

(a) Although it is important to model subjects' SRH status outcome, modelling the markers in a univariate set-up may be problematic. Researchers (Liang *et al.*, 2007; Bourne, 2010; Bond *et al.*, 2006; Santiago *et al.*, 2010) have raised some concerns about the reliability of different markers used for SRH measures for the following reasons.
   (i) FS and SRH are subject to day-to-day fluctuations.
   (ii) For FS, subjects were asked 'Do you have difficulty (with a specific Activity of Daily Living (ADL) or Instrumental Activity of Daily Living (IADL))?'. The questions as shown in Table 2 might not make clear what level of performance is required in each subject's perspective to meet particular criteria.
   (iii) People's perception may inflate or deflate their status and the subjective indices in measuring health status would open themselves to systematic and unsystematic biases.

   As a consequence analysing a single response like FS or SRH alone to understand health status can be subject to daily fluctuations, calling into question the reliability of the response. This reliability issue may not suffice in understanding the underlying health status of the elderly. To overcome this issue and to estimate health status accurately and coherently, one should ideally model the trajectories of the multiple responses simultaneously. To mitigate the variability in the single response, we thus propose to model FS and SRH jointly.

(b) It is well known that the repeatedly measured events contributed by the same subjects tend to be correlated and a current event in our scenario is likely to be associated with a past event. In this perspective, we consider that SRH depends on the latent health status, where the hypothesized unobserved states of health status follow a multistate hidden Markov model. The advantage of the latent variable approach is that it can better handle the measurement error that is inherent in self-reported outcomes and accommodates the

Table 2. Categories of ADL and IADL used in the LSOA II study

ADL categories	*IADL categories*
Bathing or showering; dressing; eating; getting in and out of bed or chairs; walking; using a toilet	Preparing meals; shopping for groceries; managing money; using the telephone; doing heavy housework; doing light housework; getting outside; managing medication

variation in SRH. Additionally, we assumed a random-effect structure to account for the heterogeneity across the subjects.

(c) FS can be analysed by the ordered logistic regression model, which is one of the widely used approaches to estimating an ordinal response (Agresti, 2007). In this paper, we further extend the ordered logistic model by embedding smoothing splines to offer a greater flexibility. Previous research has shown that FS relies on age (Hubbard *et al.*, 2009), but not necessarily with a linear dependence. In the absence of information on the true functional form of the age covariate, we use smoothing splines to model the age effect on FS.

(d) Another complicating aspect of a study with older individuals is that researchers often confront high dropout rates due to death or some other unknown reasons. Decedents and losses to follow-up were relatively high (32%) in the LSOA II study. Since it was noted that the respondents who dropped out of a survey between waves were more likely to be disabled (Lee, 2000), these dropouts cannot be regarded as missing at random. Although many previous studies in research into aging have assumed missingness at random, their methods are generally inappropriate as it would result in a loss of precision and induce a bias in parameter estimates. Instead, in this study, we assume that the dropouts are informative of the current health status of a subject and choose to deal with the dropouts in a repeated ordinal response data setting. The reason for dropout can also be associated with the health status of subjects. We address these issues by developing a multinomial logit model that allows for the interdependence of possible dropouts to various risks while correlating them through the correlation of the associated unobserved heterogeneities. Our data reveal that a person whose FS is severe in wave 1 or wave 2 is more likely to die in the follow-up wave. This leads us to believe that it is important to adjust the dropout. We have explored the descriptive statistics between obese people who dropped out and those who did not drop out, and it is shown in Table 3. A comparison of these two groups suggests that the sample characteristics of each group are sufficiently different, thus supporting our hypothesis that we need to account for the probability of dropout.

However, each of the above complications does not arise from a distinct or separate setting in the research into aging. To understand the whole dynamics of ordinal SRH outcomes better, it is then necessary to develop a unified statistical model addressing these issues. Although the joint model that we propose in this paper is generally suited for use in health and disease research, where the ordered value responses are observed at successive time points, we further extend it by addressing some of the challenges by incorporating

(a) the semiparametric features into the ordinal logistic model,
(b) modelling the underlying latent states of health that are associated with SRH,

Table 3. Descriptive statistics between obese people who dropped out and those who did not drop out

Variable	*Results for dropout*	*Results for no dropout*
FS at wave 1	1	2
FS at wave 2	3	3
FS at wave 3	3	3
Male (*versus* female)	30.57%	36.42%
White (*versus* non-white)	83.65%	79.96%
Age	75.46	76.73
Unhealthy (*versus* healthy) SRH at wave 1	29.51%	41.81%
Unhealthy (*versus* healthy) SRH at wave 2	37.58%	46.6%
Unhealthy (*versus* healthy) SRH at wave 3	41.61%	65.42%
High school education or beyond (*versus* lower than high school education)	71.5%	63.58%
Number of conditions $\geqslant 3$ (*versus* number of conditions < 3) at wave 1	36.94%	38.36%
Number of conditions $\geqslant 3$ (*versus* number of conditions < 3) at wave 2	46.28%	41.36%
Number of conditions $\geqslant 3$ (*versus* number of conditions < 3) at wave 3	62.42%	72.28%

(c) jointly modelling the bivariate ordinal outcomes to mitigate the variability of the single response and
(d) accounting for the non-ignorable missing data due to different reasons through a multinomial logit model.

In essence, our final model has a semiparametric component for the accommodation of a potentially non-linear aging effect, the drop-out components and Markov process; all these components are embedded in the bivariate ordinal regression through the shared latent random effects.

1.2. Variables

Our interest in this study is the obese group. We calculated the body mass index BMI (i.e. the weight in kilograms divided by height in metres squared) and used the standard cut point BMI$\geqslant$30 to classify obese individuals. Individuals with BMI over 55 were excluded because such extreme values are rare. In all there were 9447 subjects, of whom 1267 people were categorized in the obese group. We further excluded 20 subjects who were institutionalized because they could not provide sufficient information for the FS measure that evaluates how the subject interacts with, at least partially, the surrounding environment. Among 1248 obese individuals, 332 had incomplete data by not providing a valid answer (e.g. did not know, refused or other responses). Finally, 936 obese subjects were analysed for this paper. As a comparison with the obese group, 3070 individuals in the normal weight group ($18.5 \leqslant$ BMI $\leqslant 24.9$) were also analysed.

We consider two response variables: FS and SRH. However, note that FS and SRH were also used as covariates with other variables for modelling the dropouts as these may have valuable information on whether a subject drops out. FS is an ordinal variable and is categorized accord-

Table 4. Descriptions of variables and reference for the analysis of the LSOA II data

Variable	*Description*
Dependent variables	
FS	FS:
	1, independent without any ADL or IADL disability
	2, IADL disabled only
	3, moderately ADL disabled (1 or 2 ADLs impaired)
	4, severely ADL disabled ($\geqslant$ 3 ADLs impaired)
SRH	SRH:
	1, excellent
	2, very good
	3, good
	4, fair
	5, poor
Covariates	
RACE	0, non-white†
	1, white
EDU	0, low education (less than high school)†
	1, high education (at least high school)
COND	0, good condition (fewer than 3 chronic conditions)†
	1, bad condition (3 or more chronic conditions)
SEX	0, female†
	1, male
AGE	Base age of the respondent (continuous, 70–99 years)
TIME	Time between two consecutive waves (continuous)

†Reference variable.

ing to the ADL and IADL items in the LSOA II study. More specifically, the four levels of FS are 1, independent without any ADL or IADL disability, 2, IADL disabled only, 3, moderately ADL disabled (one or two ADLs impaired), and 4, severely ADL disabled (three or more ADLs impaired). This classification has been used in several previous studies of FS in older adults (Mor *et al.*, 1994; Anderson *et al.*, 1998). In constrast, SRH was assessed by asking 'Would you say your health in general is excellent, very good, good, fair, or poor?'. Thus, SRH is a five-point scaled ordered value: excellent, very good, good, fair and poor.

The covariates that were used in this analysis are listed in Table 4. The vector of baseline characteristics is $B_i = (\text{AGE}, \text{SEX}, \text{RACE}, \text{EDU})$, where AGE, SEX, RACE and EDU are respectively the ith subject's age at the start of the interview in 1994, gender, race (white *versus* non-white) and the level of education (completion of 16 years or less *versus* completion of more than 16 years). The vector of the time varying covariates is $T_i = (\text{TIME}, \text{COND})$, where TIME is a time difference between two measurements (the waves of the LSOA II study have non-uniform time lags (from about 1.2 to 3 years) between each follow-up period), and COND is a chronic condition diagnosed by doctors such as diabetes, arthritis, heart disease, stroke, cancer, hypertension and asthma (1 if the number is greater than 3, and 0 otherwise). The variables that were selected for this study are mostly adapted from Ten Have *et al.* (2000), Lee (2000) and Anderson *et al.* (1998). However, it is worth noting that those variables are not the only important predictors to model health status. More review on risk factors for the health status of the elderly can be found in Stuck *et al.* (1999).

There have been a large number (32%) of the subjects in the LSOA II data who dropped out during a long follow-up. To incorporate a non-ignorable missing data mechanism, we classified

the missingness status of each subject into three categories: observed $\mathcal{O}$, death, $\mathcal{D}$, and unknown reasons, $\mathcal{U}$.

2. Joint statistical model

In this section, we describe our proposed joint statistical model. Our joint model consists of three main components: an ordinal logistic model for the dependent variable SRH along with a two-state hidden Markov model, another ordinal logistic model for dependent variable FS and a discrete competing risk model for the dropout process. Further, all three components are joined by correlating the individual random effects from each component resulting in the joint model. This correlated random-effects structure offers an appealing framework for the joint modelling of the three outcomes and induces the dependence between them.

2.1. Modelling self-rated health

Let SRH_{it} be SRH for the ith subject at the tth time. Since the SRH variable is ordinal, and usually an ordinal outcome does not have a natural distribution, we assume that the observed ordinal response SRH_{it} is generated from an underlying latent variable SRH^*_{it} with a set of threshold values $\delta = (\delta_1, \ldots, \delta_K)^{\mathrm{T}}$. Specifically, SRH_{it} falls in category k, if the SRH^*_{it} of the latent response exceeds δ_{k-1} but does not exceed δ_k. Hence, letting $\delta_0 = -\infty$ and $\delta_K = \infty$, the model for SRH_{it} is given by

$$\mathrm{SRH}_{it} = k \qquad \text{if } \delta_{k-1} \leqslant \mathrm{SRH}^*_{it} < \delta_k, \quad k = 1, \ldots, K, \quad i = 1, \ldots, N, \quad t = 1, \ldots, T.$$

For the cumulative events, we obtain $(\mathrm{SRH}_{it} \leqslant k) \Leftrightarrow (\mathrm{SRH}^*_{it} < \delta_k)$. The threshold values must be monotonically increasing to reflect the ordinal nature of the observed outcomes. At the second stage, we consider a mixed effects regression model for the unobserved SRH^*_{it} which is expressed as

$$\mathrm{SRH}^*_{it} = \lambda_{0i} + \lambda_1 S_{it} + \varepsilon_{it}, \tag{1}$$

where λ_{0i} is a subject-specific random intercept, λ_1 is a fixed slope coefficient, S_{it} is the unobserved health state and ε_{it} has the standard logistic distribution. We assume that S_{it} is a binary latent variable having two underlying states (healthy and unhealthy) and thus follows a two-state discrete hidden Markov model. The transition probability is modelled by a logistic regression with relevant covariates. Thus, the transition probability is further modelled as

$$S_{it} | S_{i,t-1} \sim \mathrm{Bin}\{1, \mathrm{logit}^{-1}(\mathbf{X}_{it}\boldsymbol{\xi})\}. \tag{2}$$

This formulation is important as the transition probabilities between any two states at time t are allowed to depend on the subject's state at previous time $t-1$ and to be influenced by the background independent variables $\mathbf{X}_{it}$, which can accommodate both fixed or time varying covariates. The transition probability represents the probability of transitioning to or remaining in the unhealthy state given the covariate.

2.2. Modelling functional status

Let FS_{it} be the value of FS of the ith subject at time t. Since FS_{it} is also ordinal, we follow the approach of the previous section and model it by using an ordinal regression.

Assuming $\nu = (\nu_1, \ldots, \nu_J)^{\mathrm{T}}$ as the set of threshold values and letting $\nu_0 = -\infty$ and $\nu_J = \infty$ the model for FS_{it} is given by

$$\mathrm{FS}_{it} = j \qquad \text{if } \nu_{j-1} \leqslant \mathrm{FS}^*_{it} < \nu_j, \quad j = 1, \ldots, J, \quad i = 1, \ldots, N, \quad t = 1, \ldots, T,$$

$$\mathrm{FS}^*_{it} = \zeta_i + f_{\mathrm{female}_i}(\mathrm{AGE_i}) + f_{\mathrm{male}_i}(\mathrm{AGE_i}) + \mathbf{W}_{it}\boldsymbol{\beta} + \varepsilon'_{it}, \tag{3}$$

where FS^*_{it} is modelled as a partial linear model, where the effect of aging is modelled through a spline function varying by gender. ζ_i is the random effect, $\mathbf{W}_{it}$ are the fixed or time varying covariates, $\boldsymbol{\beta}$ is a vector of regression coefficients associated with $\mathbf{W}_{it}$, and ε'_{it} has a standard logistic distribution. Although S_{it}, the unobserved health status of the ith individual at time t, was only considered for modelling SRH, it is also an important predictor for FS. However, we do not include S_{it} in model (3) for the following reasons. When S_{it} is already considered in model (1) the additional latent variable S_{it} in model (3) might bring an identification issue. In addition, model (3) accounts for a random effect as well as a non-linear effect. Thus, the meaning of S_{it} in model (3) would not be the same as in model (1). Finally, FS* is also influenced by SRH* as FS* and SRH* are jointly modelled by their random effects. Therefore, introducing S_{it} in model (3) would not be necessary.

In this application, the effect of AGE on FS may not be linear and can also vary across gender. Thus, the AGE effect on FS is modelled by an unspecified non-parametric function across gender. Following Ruppert *et al*. (2003), we assume that the functions are modelled as a spline function which takes the following general form of a piecewise polynomial of degree l:

$$f(\mathrm{AGE}_i) = b_1\,\mathrm{AGE}_i + \ldots + b_l\,\mathrm{AGE}_i^l + \sum_{d=1}^{D} \omega_d^l (\mathrm{AGE}_i - \kappa_d)_+^l,$$

where $X_+ = X$ if $x > 0$, and $X_+ = 0$ otherwise, and κ_d are known knot points. The choice of the knots will be described in Section 3. $(b_1, \ldots, b_l)$ is the vector of regression coefficients, and ω_d are considered to be the random parameters which are usually assumed to have an $N(0, \sigma_\omega^2)$ distribution. Note that, in the above formulation of the spline, to avoid identifiability we do not include an intercept. For practical computational purposes the order of the spline is taken as 1. We also experimented with an order 2 spline but did not find any changes in the shape of the curve.

2.3. Modelling the competing risk

If the dropouts are due to a mechanism that is unrelated to the investigations, i.e. the unobserved behaviours are missing completely at random, these dropouts can be ignored. However, it is unlikely to be so for most of the longitudinal studies on older adults since the reason for dropouts such as unknown loss to follow-up and death may be due to underlying longitudinal health outcomes, such as FS and SRH. Therefore, two dropout reasons, 'death' and 'unknown', are also considered in the model. We define them as unknown if subjects did not participate in the survey and had not come back by the end of the study.

Specifically, we define the competing risk indicator as R_{it} taking three values depending on the missing data status as follows:

$$R_{it} = \begin{cases} \mathcal{O} & \text{if subject } i \text{ is observed at measurement } t, \\ \mathcal{D} & \text{if subject } i \text{ is dead at measurement } t, \\ \mathcal{U} & \text{if subject } i \text{ is an unknown loss to follow-up at measurement } t. \end{cases}$$

We assume that the outcome measurement at $t = 1$ is observed for all subjects, and death and unknown are absorbing states, i.e., once a subject has dropped out or died, we shall no longer observe measurements for this subject. Particularly, the 'missingness' mechanism is modelled with a multinomial logit regression to calculate the transition probabilities of the missingness indicators as follows:

$$\pi_{it}^{(r)} = \Pr(R_{it} = r | R_{i,t-1} = \mathcal{O}) = \frac{\exp(\mathbf{Z}_{it}^{\mathrm{T}} \boldsymbol{\rho}^{(r)} + b_i^{(r)})}{\sum_r \exp(\mathbf{Z}_{it}^{\mathrm{T}} \boldsymbol{\rho}^{(r)} + b_i^{(r)})}, \qquad r = \mathcal{O}, \mathcal{D}, \mathcal{U}, \tag{4}$$

where $b_i^{(r)}$ is a random effect of subject i for missingness reason r, $\mathbf{Z}_{it}$ is a vector of covariates and $\boldsymbol{\rho}^{(r)}$ is its associated regression parameter.

2.4. Joint model and the likelihood

To estimate FS and SRH jointly, we combine the techniques of hidden Markov models and competing risk models. The association between FS_{it}, SRH_{it}, S_{it} and R_{it} is modelled by the assumption that the random effects $(\zeta_i, \lambda_{0i}, b_i^{(\mathcal{D})}, b_i^{(\mathcal{U})})$ jointly have a multivariate normal distribution:

$$a_i = (\zeta_i, \lambda_{0i}, b_i^{(\mathcal{D})}, b_i^{(\mathcal{U})})^{\mathrm{T}} \sim \mathrm{MVN}(\mathbf{0}_4, \boldsymbol{\Sigma}_{4\times 4}), \tag{5}$$

where $\boldsymbol{\Sigma}$ is the variance–covariance matrix of the vector a_i.

Let Ω_1 be the set of parameters from the model of SRH, Ω_2 be the set of parameters from the model for FS and Ω_3 be the set of parameters from the competing risk model. Let $\Omega = (\Omega_1, \Omega_2, \Omega_3)$. Then, the likelihood for the observed data for the ith individual conditional on Ω and the random effects a_i is proportional to

$$L_i(\mathrm{SRH}_{it}, \mathrm{FS}_{it}, R_{it} | \Omega, a_i) \propto L_i(\mathrm{SRH}_{it} | \Omega_1, a_i)\, L_i(\mathrm{FS}_{it} | \Omega_2, a_i)\, L_i(R_{it} | \Omega_3, a_i)\, f(a_i | \Sigma),$$

where

$$L_i(\mathrm{SRH}_{it} | \Omega_1, a_i) \propto \prod_t \prod_k \Pr(\mathrm{SRH}_{it} = k | \Omega_1, a_i)^{I(\mathrm{SRH}_{it}=k)},$$

$$\Pr(\mathrm{SRH}_{it} = k) = \Pr(\delta_{k-1} \leqslant \mathrm{SRH}_{it} < \delta_k | \Omega_1, a_i),$$

where $I(\cdot)$ is an indicator variable, and

$$L_i(\mathrm{FS}_{it} | \Omega_2, a_i) \propto \prod_t \prod_j \Pr(\mathrm{FS}_{it} = j | \Omega_2, a_i)^{I(\mathrm{FS}_{it}=j)},$$

$$\Pr(\mathrm{FS}_{it} = j) = \Pr(\nu_{j-1} \leqslant \mathrm{FS}_{it} < \nu_j | \Omega_2, a_i),$$

and

$$L_i(R_{it} | \Omega_3, a_i) \propto \prod_t \prod_r d_{itr} \pi_{it}^{(r)}, \qquad r = \mathcal{O}, \mathcal{D}, \mathcal{U},$$

where $d_{itr} = 1$ if subject i drops out for reason r at time t, and otherwise $d_{itr} = 0$. Finally, $f(a_i | \Sigma)$ is the multivariate normal distribution for the random effects a_i.

3. Data analysis

3.1. Model specification

We analysed the LSOA II data by using the model that was described in the previous section. We denote $\mathrm{FS}_{it}^{(\mathrm{O})}$ $(i = 1, \ldots, 935, t = 1, 2, 3)$ as the FS of the obese subjects, and $\mathrm{FS}_{it}^{(\mathrm{N})}$ $(i = 1, \ldots, 3070; t = 1, 2, 3)$ as the normal weight subjects.

We assume that the observed SRH_{it} is determined by four cut-off points and the observed FS_{it} is determined by three cut-off points. For the covariates in equation (2), we assume that

$$\mathbf{X}_{it}\boldsymbol{\xi} = (\xi_1 + \xi_2 \mathrm{RACE}_i + \xi_3 \mathrm{EDU}_i + \xi_4 \mathrm{SEX}_i + \xi_5 \mathrm{AGE}_i + \xi_6 \mathrm{COND}_{it} + \xi_7 \mathrm{TIME}_{it}).$$

For equation (3), we take the covariates

$$\begin{aligned} \mathrm{FS}^*_{it} = \zeta_i + \beta_1 \mathrm{RACE}_i + \beta_2 \mathrm{EDU}_i + \beta_3 \mathrm{COND}_{it} + \beta_4 \mathrm{TIME}_{it} \\ + f_{\mathrm{female}_i}(\mathrm{AGE}_i) + f_{\mathrm{male}_i}(\mathrm{AGE}_i) + \varepsilon'_{it}, \end{aligned}$$

where $f_{\mathrm{SEX}_i}(\mathrm{AGE}_i)$ is fitted by a P-spline with eight equally spaced knots.

For the competing risk models in equation (4), we take the covariates

$$\mathbf{Z}_{it} = (1, \mathrm{FS}_{i,t-1}, \mathrm{COND}_{i,t-1}, \mathrm{SRH}_i, \mathrm{SEX}_i, \mathrm{RACE}_i, \mathrm{AGE}_i, \mathrm{EDU}_i)^{\mathrm{T}}.$$

Finally, the joint model of FS* and SRH*, and the competing risk models are connected by random effects $a = (\zeta_i, \lambda_{0i}, b_i^{(\mathcal{D})}, b_i^{(\mathcal{U})}) \sim \mathrm{MVN}(\boldsymbol{\mu}_4, \boldsymbol{\Sigma}_{4\times 4})$. To complete the Bayesian specification we assign a weakly informative conjugate prior to the parameters to obtain well-defined posteriors. Prior distributions were centred near zero as we were uncertain about the parameter values. For each fixed effect, we assumed a normal density prior. Specifically, we assigned a weakly informative independent and identically distributed $N(0, \text{precision} = 0.25)$ prior. Since our model has a logistic regression structure, a prior variance of 4 (a precision of 0.25) implies that the odds ratio has a mean of 1 and the 95% spread is between exp(−4) and exp(4). This is a very wide range of prior guess in terms of odds ratio and is reasonably informative (Dunson *et al*., 2003). For the variance parameter, we used an inverse gamma IG(2.01, 1.01) prior, such that the prior mean is 1 and prior variance is 100, whereas for the variance–covariance matrix we assumed an inverse Wishart prior with 5 degrees of freedom.

The posterior distributions are analytically intractable. However, the models described previously can be fitted by using Markov chain Monte Carlo methods such as the Gibbs sampler (Gelfand and Smith, 1990). Since the full conditional distributions are not standard, a straightforward implementation of the Gibbs sampler using standard sampling techniques may not be possible. Sampling methods can also be performed by using the Metropolis–Hastings algorithm. Samples were directly obtained from the joint posterior distribution of the parameters as well as the latent variables. Implementation of this method is relatively easy and done in the publicly available software WinBUGS (Spiegelhalter *et al*., 2003). The samples from the posterior that were obtained from the Markov chain Monte Carlo algorithm will allow us to achieve summary measures of the parameter estimates and to obtain credible intervals of the parameters of interest.

We ran two chains of the Gibbs sampler with widely dispersed initial values. The initial values for the fixed parameters were selected by starting with the prior mean and covering ±3 standard deviations. The initial values for the precision were arbitrarily selected. We also centred the covariates about the mean to have better convergence. In our simulation, 10000 samples were discarded as burn-in and of the next 40000 samples we used every sixth value to construct the posterior estimate. Convergence was assessed visually by monitoring the dynamic traces of Gibbs iterations and by computing the Gelman–Rubin convergence statistic (Gelman and Rubin, 1992). To check for sensitivity, we ran the proposed model with different sets of priors and found little evidence of any prior sensitivity, although slow mixing was evident in analyses using a highly diffuse prior.

3.2. Analytical results

To investigate the longitudinal process of health status attributed to obesity, we applied the LSOA II data to the model proposed in Section 2. Note that we used the term 'normal' to refer to the elderly group with the normal weighted group and the term 'obese' as the obese elderly group. All the tables in this section report the posterior mean along with the 95% credible

Table 5. Parameter estimates of modelling FS, with credible intervals

Parameter	*Estimates for the normal group*		*Estimates for the obese group*	
	Mean	*95% credible interval*	*Mean*	*95% credible interval*
β_1 (RACE)	−0.60	(−0.97, −0.26)	0.05	(−0.41, 0.50)
β_2 (EDU)	−0.56	(−0.82, −0.30)	−0.29	(−0.69, 0.08)
β_3 (COND)	1.32	(1.16, 1.48)	1.23	(0.97, 1.48)
β_4 (TIME)	0.54	(0.47, 0.61)	0.47	(0.36, 0.57)

interval. The parameter estimates for parametric parts of modelling FS shown in model (6) are listed in Table 5.

(a) We failed to find enough evidence that non-white status was related to the incidence of severe FS among the obese elderly ($\beta_1^{(O)} = -0.05$ $(-0.41, 0.50)$). However, the results suggest that severe FS among the normal elderly is prevalent in non-white compared with white people ($\beta_1^{(N)} = -0.60\,(-0.97, -0.26)$).
(b) There is a negative effect of lower education on FS for both obese and normal groups ($\beta_2^{(N)} = -0.56$ $(-0.82, -0.30)$, $\beta_2^{(O)} = -0.29$ $(-0.69, -0.08)$). It might imply that the deterioration in FS may be delayed by a high level of education associated with high income and good lifestyle habits.
(c) FS is strongly accelerated by the onset of multiple chronic diseases (three or greater) ($\beta_3^{(N)} = 1.32$ (1.16, 1.48); $\beta_3^{(O)} = 1.23$ (1.00, 1.48)).
(d) The time effect is also statistically significant ($\beta_4^{(N)} = 0.54$ (0.47, 0.61); $\beta_4^{(O)} = 0.47$ (0.36, 0.57)), indicating that FS declined in nature as time went on without any active treatments.

Little is known about the gender difference in the level of FS across age, particularly for the obese elderly population. Our analysis results show that, with advancing age, FS grows worse in both men and women. For the normal BMI group, the distribution did not differ between male and female subjects, indicating that the aging effects were similar for males and females. However, FS is greatly affected in obese women compared with obese men at their later ages with the highest level at 85 years and older. This confirmed the previous findings in Himes (2000) where obesity was related more strongly to limitations in physical activity for women. Since women have a longer average lifespan than men, it is possible that obese widows who live alone have higher levels of malnutrition, which can lead to poorer FS.

Next, we considered the transition probabilities to transit or remain in the unhealthy state. Table 6 displays the log-odds ratios and their 95% credible interval for the covariates that were used in modelling the transition probabilities. In general, the directions of the coefficients were the same for both the normal and the obese elderly groups.

(a) White normal elderly people were less likely to remain or transfer to the unhealthy state compared with non-white normal BMI elderly people. However, the race effect for the obese group was not statistically significant ($\xi_1^{(N)} = -2.27\,(-3.39, -1.38)$; $\xi_1^{(O)} = -2.10\,(-5.46, 1.30)$). Higher education was associated with the transition probability to the unhealthy state for the normal group, but evidence for the obese elderly was not sufficient ($\xi_2^{(N)} = -2.46$ $(-3.56, -1.57)$; $\xi_2^{(O)} = -3.65$ $(-8.01, 0.09)$).

Table 6. Log-odds ratios and 95% credible intervals of remaining in or transitioning to the unhealthy state obtained from fitting the hidden Markov model

Parameter	*Results for the normal group*		*Results for the obese group*	
	Mean	*95% credible interval*	*Mean*	*95% credible interval*
ξ_1 (RACE)	−2.27	(−3.39, −1.38)	−2.10	(−5.46, 1.30)
ξ_2 (EDU)	−2.46	(−3.56, −1.57)	−3.65	(−8.01, 0.09)
ξ_3 (SEX)	0.82	(0.36, 1.35)	−2.70	(−5.06, 0.22)
ξ_4 (AGE)	0.06	(0.02, 0.09)	0.07	(−0.13, 0.33)
ξ_5 (COND)	3.59	(2.66, 4.80)	12.19	(8.70, 15.50)
ξ_6 (TIME)	0.39	(0.19, 0.62)	2.44	(1.09, 3.89)

(b) Normal BMI men had a higher probability of making a transition to the unhealthy state compared with normal BMI women ($\xi_3^{(N)} = 0.82\,(0.36, 1.35)$; $\xi_3^{(O)} = -2.70\,(-5.06, 0.22)$). This can be attributed to the fact that men are more likely to suffer from severe chronic conditions and fatal diseases than women are.

(c) Aging was not clearly associated with an increased probability of making a transition into the unhealthy state for the obese elderly ($\xi_4^{(N)} = 0.06\ (0.02, 0.09)$; $\xi_4^{(O)} = 0.07\,(-0.13, 0.33)$).

(d) Multiple chronic conditions had the strongest odds of making a transition to or remaining in the unhealthy state ($\xi_5^{(N)} = 3.59\,(2.66, 4.80)$; $\xi_5^{(O)} = 12.19\,(8.70, 15.50)$).

(e) The time effect had a strong association with a transition or remaining in the unhealthy state ($\xi_6^{(N)} = 0.39\,(0.19, 0.67)$; $\xi_6^{(O)} = 2.44\,(1.09, 3.89)$).

The parameter estimates for the competing risk model are reported in Table 7. From the estimates of parameters in the competing risk models, we made the following observations.

(a) The dropout probability due to death in obese elderly people was related to both SRH and FS ($\rho_{\mathcal{D},1}^{(O)} = 0.53\,(0.31, 0.74)$; $\rho_{\mathcal{D},3}^{(O)} = 0.49\,(0.77, 0.74)$). This result supports the previous findings that SRH and FS are predictors of mortality for the obese elderly. Additionally, this result corroborates our assumption that the missing information was not missing completely at random. In other words, the probability of dropout was related to an unobserved outcome at the time of dropout. Thus, it provided justification for accounting for a dropout reason in modelling the health status of the obese elderly.

(b) We observed that multiple chronic conditions were not an important predictor for death from unknown reasons ($\rho_{\mathcal{D},2}^{(O)} = -0.30\,(-0.65, 0.06)$; $\rho_{\mathcal{U},2}^{(O)} = -0.48\,(-0.82, -0.14)$). In our data a strong relationship between FS and COND was shown for the obese elderly; at wave 3, the proportion of the poor FS (FS $\geqslant 3$) for subjects with multiple chronic conditions was nearly 50%, whereas it was only 15% for less than one chronic condition. Compared with the normal group, 23% of subjects with multiple conditions and 16% of subjects with fewer than two conditions had poor FS. Therefore, COND should be interpreted carefully owing to the possible multicollinearity between FS and COND.

(c) Men had a higher probability of death. We did not find sufficient evidence that the dropouts due to unknown reasons were more common among men ($\rho_{\mathcal{D},4}^{(N)} = 0.68\,(0.52, 0.85)$; $\rho_{\mathcal{D},4}^{(O)} = 0.67\ (0.30,1.03)$).

Table 7. Parameter estimates of the competing risk model

Parameter	*Results for the normal group*		*Results for the obese group*	
	Mean	*95% credible interval*	*Mean*	*95% credible interval*
Death				
$\rho_{\mathcal{D},0}$ (intercept)	−10.16	(−11.57, −8.86)	−10.79	(−13.13,−8.12)
$\rho_{\mathcal{D},1}$ (FS)	0.50	(0.36, 0.68)	0.53	(0.31, 0.74)
$\rho_{\mathcal{D},2}$ (COND)	0.01	(−0.19, 0.21)	−0.30	(−0.65, 0.06)
$\rho_{\mathcal{D},3}$ (SRH)	0.28	(0.14, 0.41)	0.49	(0.27, 0.74)
$\rho_{\mathcal{D},4}$ (SEX)	0.68	(0.52, 0.85)	0.67	(0.30, 1.03)
$\rho_{\mathcal{D},5}$ (RACE)	−0.01	(−0.32, 0.29)	−0.18	(−0.59, 0.27)
$\rho_{\mathcal{D},6}$ (AGE)	0.08	(0.07, 0.10)	0.07	(0.04, 0.10)
$\rho_{\mathcal{D},7}$ (EDU)	−0.09	(−0.29, 0.11)	0.06	(−0.31, 0.42)
Unknown				
$\rho_{\mathcal{U},0}$ (intercept)	−2.85	(−4.50, −1.18)	−1.67	(−4.05, 1.14)
$\rho_{\mathcal{U},1}$ (FS)	−0.04	(−0.18, 0.14)	−0.10	(−0.36, 0.17)
$\rho_{\mathcal{U},2}$ (COND)	−0.18	(−0.42, 0.05)	−0.48	(−0.82, −0.14)
$\rho_{\mathcal{U},3}$ (SRH)	0.19	(−0.03, 0.44)	0.01	(−0.32, 0.31)
$\rho_{\mathcal{U},4}$ (SEX)	−0.02	(−0.21, 0.16)	0.03	(−0.31, 0.35)
$\rho_{\mathcal{U},5}$ (RACE)	−0.37	(−0.67, −0.05)	−0.31	(−0.68, 0.07)
$\rho_{\mathcal{U},6}$ (AGE)	0.01	(0.00, 0.03)	0.01	(−0.02, 0.04)
$\rho_{\mathcal{U},7}$ (EDU)	−0.29	(−0.51, −0.05)	−0.19	(−0.50, 0.15)

(d) The race effect for the obese elderly was not statistically significant. This is in contrast with the case of the normal BMI group, in which white people were less likely to drop out for unknown reasons ($\rho^{(N)}_{\mathcal{D},5} = -0.01\,(-0.32, 0.29)$; $\rho^{(N)}_{\mathcal{U},5} = -0.18\,(-0.59, 0.27)$; $\rho^{(O)}_{\mathcal{D},5} = -0.37\,(-0.67, -0.05)$; $\rho^{(O)}_{\mathcal{U},5} = -0.31\,(-0.68, 0.07)$).

(e) As expected, older subjects were more likely to drop out because of death. The estimates of the parameters $\rho^{(N)}_{\mathcal{D},6}$ and $\rho^{(O)}_{\mathcal{D},6}$ were 0.08 (0.07, 0.10) and 0.07 (0.04, 0.10) respectively. However, age was not particularly related to dropout for unknown reasons ($\rho^{(N)}_{\mathcal{U},6} = 0.01\,(-0.01, 0.03)$; $\rho^{(O)}_{\mathcal{U},6} = 0.01\,(-0.02, 0.04)$).

(f) Education was not associated with dropout reasons for the obese elderly; however, higher education was negatively related to the probability of dropout due to unknown reasons for the normal BMI group ($\rho^{(N)}_{\mathcal{D},7} = -0.09\,(-0.29, 0.11)$; $\rho^{(O)}_{\mathcal{D},7} = 0.06\,(-0.31, 0.42)$; $\rho^{(N)}_{\mathcal{U},7} = -0.29\,(-0.51, -0.05)$; $\rho^{(O)}_{\mathcal{U},7} = -0.19\,(-0.50, 0.15)$).

3.3. Model comparison

To compare our proposed model, we compute the deviance information criterion (DIC) (Spiegelhalter *et al.*, 2002). The DIC is a Bayesian generalization of the Akaike information criterion and it trades off a measure of model adequacy against a measure of complexity. It is based on the posterior distribution of the log-likelihood or deviance. The DIC uses a measure of complexity for the effective number of parameters that is based on an information theoretic argument. This quantity is readily obtained from a Markov chain Monte Carlo analysis and is in built into the WinBUGS software.

We compare our proposed joint model with an independent model, i.e. none of the three components (SRH, FS and dropout) are correlated. We also compare both the model for the

obese and the model for the normal weight elderly. The DIC for our proposed joint model was 15955.4 and 128921.8 for the obese and normal weight elderly respectively. The DIC for the independent model was 23003.7 and 160382.9 for the obese and normal weight elderly respectively. Thus, on the basis of the DIC values, the joint model was a better model for the data.

4. Discussion and future research

Though many studies have reported on the health status of the elderly, few researchers have conducted modelling of health status focusing on the obese elderly. Using data from the LSOA II study we developed a joint model which accounts for the dropout, combined different indices of health status and allowed for non-linear effects of covariates on the response. One strength of the present study is the long-term evaluation of health status of the obese elderly compared with normal BMI elderly people. A further strength is the comprehensive evaluation of a broad spectrum of the factors to influence health status by taking those complications into account.

For instance, higher educational levels positively affected FS and are related to higher transition probabilities to the healthy state. Differences in education may contribute to disparities in health-related behaviour patterns (e.g. diet and exercise) affecting health outcomes. Next, our results show that males were more likely to remain in or to transition to the unhealthy state compared with females, which might be related to the fact that men are more likely to suffer from severe chronic conditions and fatal diseases than women. Since multiple chronic diseases also had a significant influence on health outcome, a community-based chronic disease self-management programme will be beneficial (Lorig *et al.*, 2001). However, we demonstrated that women over 85 years old had more problems in physical functions than their counterparts.

Our findings underscore the similarities and dissimilarities between the normal weight and obese groups. The transition probabilities involving the hidden Markov process of latent health status between the two groups were similar. However, the estimation of parameters in our model indicated that some factors including race or ethnicity, age and SRH have different effects compared with the normal weight group, suggesting that those two groups are not identical, and different approaches for the obese group are needed for effective management of obesity. In addition, we observed that the probability of dropout was associated with the previous status of the health outcome and SRH, confirming our belief that the missing information is non-ignorable.

Despite the success of our proposed method in providing useful information for the health consequences of the obese elderly, we acknowledge that some limitations in modelling are inherent in the incomplete information of the LSOA II data and the model complexity. Our definition of both FS and SRH in this paper encompasses only the severity or intensity. However, it can be extended to include more responses, such as frequency, duration or effect of the symptom. The subsequent interviews in the LSOA II study were about 2 years apart. Because of the episodic and recurrent nature of poor health, it is highly possible that multiple changes in FS and/or SRH occur between time points t and $t+1$. Therefore, taking account of the number of change in FS and/or SRH between two time points may influence the current FS and/or SRH of the elderly, which is worthy of further investigation.

Finally, we remark that the complicated nature of the data made our proposed model complex. However, our model is inspired by considerations of utility and value of the information that is provided and is required to seek advancement of analysing the longitudinal health status of the elderly. Furthermore, the situations that we considered in this analysis are commonly encountered in the observational study as well as often found in behavioural and clinical

research. We believe that our unified model can be applied to such data and potentially enrich the results.

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