# Mortality Rate Estimation and Standardization for Public Reporting: Medicare's Hospital Compare 

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## Supplemental Material

## A Appendix

## A. 1 MCMC Posterior Calculation

To fit our fully Bayesian hierarchical models (C,C), (L,C), (S,L) and (SLI,L) we use MCMC simulation sampling from the data induced posterior to calculate quantities of interest. To describe this, it will be convenient to index each of our hierarchical models by $(\boldsymbol{\alpha}, \boldsymbol{\beta}, \boldsymbol{\psi})$, where $\boldsymbol{\alpha}=$ $\left(\alpha_{1}, \ldots, \alpha_{H}\right)^{\prime}$ denotes the $H$ hospital effects, $\boldsymbol{\beta}$ denotes the individual fixed effect coefficients, and $\boldsymbol{\psi}$ denotes $\sigma_{\beta}^{2}$ and all other hyperparameters associated with $\mu_{h}(\boldsymbol{z})$ and $\sigma_{h}^{2}(\boldsymbol{z})$.

We use the Gibbs sampler to simulate from $\pi(\boldsymbol{\alpha}, \boldsymbol{\beta}, \boldsymbol{\psi} \mid \boldsymbol{Y})$. In principle, this would be obtained by successive substitution sampling from the full conditionals, Casella and George (1992). However, because $\pi(\boldsymbol{\alpha} \mid \boldsymbol{Y}, \boldsymbol{\beta}, \boldsymbol{\psi})$ and $\boldsymbol{p}(\boldsymbol{\beta} \mid \boldsymbol{Y}, \boldsymbol{\alpha}, \boldsymbol{\psi})$ are not available in closed form, we proceed by Gibbs sampling from an augmented posterior. Analogous to the augmentation for probit regression with normal latent variables (Albert and Chib 1993), a suitable augmentation for logistic regression is obtained with the introduction of a vector of Pólya-Gamma latent variables, $\boldsymbol{\omega}=\left\{\omega_{h j}\right\}$, one for each $h j$, to create a joint posterior $\boldsymbol{p}(\boldsymbol{\omega}, \boldsymbol{\alpha}, \boldsymbol{\beta}, \boldsymbol{\psi} \mid \boldsymbol{Y})$, (Polson, Scott and Windle 2013). The following successive substitution sampling from the full conditionals $\pi(\boldsymbol{\omega} \mid \boldsymbol{Y}, \boldsymbol{\alpha}, \boldsymbol{\beta}, \boldsymbol{\psi})$, $\pi(\boldsymbol{\alpha} \mid \boldsymbol{Y}, \boldsymbol{\omega}, \boldsymbol{\beta}, \boldsymbol{\psi}), \pi(\boldsymbol{\beta} \mid \boldsymbol{Y}, \boldsymbol{\omega}, \boldsymbol{\alpha}, \boldsymbol{\psi}), \pi(\boldsymbol{\psi} \mid \boldsymbol{Y}, \boldsymbol{\omega}, \boldsymbol{\alpha}, \boldsymbol{\beta})$, is then straightforward.

Simulation from $\pi(\boldsymbol{\omega} \mid \boldsymbol{Y}, \boldsymbol{\alpha}, \boldsymbol{\beta}, \boldsymbol{\psi})$ is obtained by simulating

$$
\begin{equation*}
\omega_{h j} \mid \boldsymbol{\alpha}, \boldsymbol{\beta} \sim \mathcal{P} \mathcal{G}\left(1, \alpha_{h}+\boldsymbol{x}_{h j}^{\prime} \boldsymbol{\beta}\right) \text { for } h=1, \ldots, H \text { and } j=1, \ldots, n_{h}, \tag{A.1}
\end{equation*}
$$

where the Pólya-Gamma distributions $\mathcal{P G}(b, c)$ are particular infinite convolutions of Gamma distributions. Polson, Scott and Windle (2013) provide a fast and exact method for simulating from any $\mathcal{P} \mathcal{G}(b, c)$ distribution, which is implemented in the R package BayesLogit (see Windle et al. (2013) for details).

To describe the simulation of $\boldsymbol{\alpha}$ and $\boldsymbol{\beta}$, let $\boldsymbol{X}$ be the complete matrix of patient attributes, $\boldsymbol{K}$ be the block diagonal matrix of hospital indicators, $\Omega=\operatorname{diag}(\boldsymbol{\omega})$ and $\boldsymbol{\kappa}=\Omega^{-1}(\boldsymbol{Y}-0.5)$. Then, simulation from $\pi(\boldsymbol{\alpha} \mid \boldsymbol{Y}, \boldsymbol{\omega}, \boldsymbol{\beta}, \boldsymbol{\psi})$ is obtained by simulating

$$
\begin{equation*}
\alpha_{h} \mid \boldsymbol{\omega}, \boldsymbol{\beta}, \boldsymbol{\psi} \sim \mathcal{N}\left(m_{\alpha_{h}}, V_{\alpha_{h}}\right) \text { for } h=1, \ldots, H, \tag{A.2}
\end{equation*}
$$

where $V_{\alpha_{h}}=\left[1 / \sigma_{h}^{2}(\boldsymbol{z})+\mathbf{1}_{v_{h}}^{\prime} \boldsymbol{\omega}_{h}\right]^{-1}$ and $m_{\alpha_{h}}=V_{\alpha_{h}}\left[\mu_{h}(\boldsymbol{z}) / \sigma_{h}^{2}(\boldsymbol{z})+\boldsymbol{\omega}_{h}^{\prime}\left(\boldsymbol{\kappa}_{h}-\boldsymbol{X}_{h} \boldsymbol{\beta}\right)\right]$.
Simulation from $\pi(\boldsymbol{\beta} \mid \boldsymbol{Y}, \boldsymbol{\omega}, \boldsymbol{\alpha}, \boldsymbol{\psi})$ is obtained by simulating

$$
\begin{equation*}
\boldsymbol{\beta} \mid \boldsymbol{\omega}, \boldsymbol{\alpha}, \boldsymbol{\psi} \sim \mathcal{N}_{d}\left(m_{\boldsymbol{\beta}}, V_{\boldsymbol{\beta}}\right) \tag{A.3}
\end{equation*}
$$

where $V_{\boldsymbol{\beta}}=\left[\left(1 / \sigma_{\boldsymbol{\beta}}^{2}\right) \boldsymbol{X}^{\prime} \boldsymbol{X}+\boldsymbol{X}^{\prime} \Omega \boldsymbol{X}\right]^{-1}$ and $m_{\boldsymbol{\beta}}=V_{\boldsymbol{\beta}}\left[\boldsymbol{X}^{\prime} \Omega(\boldsymbol{\kappa}-\boldsymbol{K} \boldsymbol{\alpha})\right]$.
Finally, simulation from $\pi(\boldsymbol{\psi} \mid \boldsymbol{Y}, \boldsymbol{\omega}, \boldsymbol{\alpha}, \boldsymbol{\beta})$, which does not depend on $\boldsymbol{\omega}$, is obtained by well known routine methods and so will not be further discussed here.

Starting with initial values, successive substitution sampling from these distributions after a suitable burn-in period and with appropriate thinning, yields a sequence

$$
\begin{equation*}
\left(\boldsymbol{\alpha}^{(1)}, \boldsymbol{\beta}^{(1)}, \boldsymbol{\psi}^{(1)}\right), \ldots,\left(\boldsymbol{\alpha}^{(S)}, \boldsymbol{\beta}^{(S)}, \boldsymbol{\psi}^{(S)}\right) \tag{A.4}
\end{equation*}
$$

which may be treated as a sample from $\pi(\boldsymbol{\alpha}, \boldsymbol{\beta}, \boldsymbol{\psi} \mid \boldsymbol{Y})$. Letting $\operatorname{logit}\left(p_{h j}\right)=\alpha_{h}+\boldsymbol{x}_{h j}^{\prime} \boldsymbol{\beta}$, the induced sequence $\boldsymbol{p}^{(1)}, \boldsymbol{p}^{(2)}, \ldots, \boldsymbol{p}^{(S)}$ will then be a sample from the induced mortality rate posterior $\pi(\boldsymbol{p} \mid \boldsymbol{Y})$. Posterior estimates of interest are obtained directly from these sequences. For example, posterior mean estimates of hospital effects are obtained by $\hat{\alpha}_{h}=\frac{1}{S} \sum_{s=1}^{S} \alpha_{h}^{(s)}$. Posterior mean estimates of individual mortality rates are obtained by $\hat{p}_{h j}=\frac{1}{S} \sum_{s=1}^{S} p_{h j}^{(s)}$. Posterior mean estimates of hospital mortality rates are obtained by $\hat{P}_{h}=\frac{1}{S} \sum_{s=1}^{S} p_{h}^{(s)}$, where $p_{h}^{(s)}=\frac{1}{n_{h}} \sum_{j=1}^{n_{h}} p_{h j}^{(s)}$. Predictive $(1-\alpha) \%$ interval bounds for these rates are obtained by the corresponding quantiles of the sampled values.

## A. 2 Modeling $\alpha_{h}$ as a Linear Function of the Number of Beds

All of our models have made use of the strong apparent relationship between hospital mortality and vol. To get further insight into the relationship between mortality rates and hospital size, we also examined the relationship between mortality rates and the hospital attribute beds2008, the number of beds in 2008, a variable that is indisputably exogenous to our observed mortality rates.

Analogous to Figure 2.1, Figure A. 1 plots the raw observed mortality rates $O_{h}$ versus beds $2008_{h}$. As summarized by the superimposed smoothing spline, the average mortality rate is decreasing as beds2008 increases. Note that many hospitals have the same value of beds2008. For example, 741 hospitals had beds2008 $=25$, which was the modal number in our data.

Figure A.2a plots the hospital effect estimates $\hat{\alpha}_{h}$ versus beds $2008_{h}$ for the (C,C) models. Just as for the plot of $\hat{\alpha}_{h}$ versus vol $_{h}$ in Figure 2.3a, the (C,C) model finds no evidence of larger hospital effects at hospitals with smaller beds $2008_{h}$. However, application of the (L,C) model with beds2008 as the single hospital attribute, again tells a dramatically different story. Just as for the plot of $\hat{\alpha}_{h}$ versus $\mathrm{vol}_{h}$ in Figure 2.3b, Figure A.2b shows that by emancipating their means
as a linear function of beds2008, the hospitals effects are dramatically higher at the hospitals with smaller beds $2008_{h}$. Thus, as opposed to the (C,C) model, the (L,C) model here will lead to systematically higher mortality rates at the smaller hospitals. Confirming that the (L,C) actually leads to improved predictions, the predictive log Bayes factor comparison, as in Section 4.1, for ( $\mathrm{L}, \mathrm{C}$ ) here vs (C,C) was 19.03, convincing evidence of a strong underlying relationship.


Figure A.1: Raw observed hospital mortality rates $O_{h}$ by beds $2008_{h}$. Average rate by beds $2008_{h}$ summarized by the green superimposed smoothing spline.


Figure A.2: $\hat{\alpha}_{h}$ vs beds2008.

## A. 3 A Second Calibration with the US News and World Report Rankings

To further illustrate our method of matched comparison in Section 4.2, we did a second calibration following the same format which we here describe briefly. The popular magazine, US News and World Report, ranks hospitals on their "heart and heart surgery" program. We repeated the observational study of low volume hospitals, replacing low volume hospitals by the top ten hospitals in the US News and World Report ranking. In the six-month validation sample, there
were 816 AMI patients in Medicare treated at the top ten hospitals in this ranking, and we matched them 5-to-1 to patients from other hospitals, checking covariate balance in parallel with Table 4.2. Unlike Table 4.2, the patients at "top ten" hospitals were not very different from all patients prior to admission. The matched comparison estimated about $2.0 \%$ lower mortality at "top ten" hospitals compared to matched controls without using the Bayes models. The Bayes models all predicted lower mortality at "top ten" hospitals than at control hospitals, but they slightly underestimated the $2.0 \%$ gain; for instance, the (SLI,L) model estimated $1.7 \%$ lower mortality at "top ten" hospitals.

## A. 4 Hospital Cross Classification by Mortality Rates

| Counts <br> $(\%)$ | (SLI,L) Low | (SLI,L) Average | (SLI,L) High | Total |
| :---: | :---: | :---: | :---: | :---: |
| (C,C) Low | 30 | 3 | 0 | 33 |
|  | $(0.68)$ | $(0.07)$ | $(0.00)$ | $(0.75)$ |
| (C,C) Average | 28 | 3289 | 1016 | 4333 |
|  | $(0.64)$ | $(78.42)$ | $(23.11)$ | $(98.57)$ |
| (C,C) High | 0 | 18 | 12 | 30 |
|  | $(0.00)$ | $(0.41)$ | $(0.27)$ | $(0.68)$ |
| Total | 58 | 3310 | 1028 | 4396 |
|  | $(1.32)$ | $(75.30)$ | $(23.28)$ |  |


| Counts <br> $(\%)$ | (SLI,L) Lower Vow | (SLI,L) Average | (SLI,L) High | Total |
| :---: | :---: | :---: | :---: | :---: |
| (C,C) Low | 0 | 0 | 0 | 0 |
|  | $(0.00)$ | $(0.00)$ | $(0.00)$ | $(0.00)$ |
| (C,C) Average | 0 | 210 | 906 | 1116 |
|  | $(0.00)$ | $(18.82)$ | $(81.18)$ | $(100.00)$ |
| (C,C) High | 0 | 0 | 0 | 0 |
|  | $(0.00)$ | $(0.00)$ | $(0.00)$ | $(0.00)$ |
| Total | 0 | 210 | 906 | 1116 |
|  | $(0.00)$ | $(18.82)$ | $(81.18)$ |  |

(c) Upper Quartile Volume Hospitals

| Counts <br> $(\%)$ | (SLI,L) Low | (SLI,L) Average | (SLI,L) High | Total |
| :---: | :---: | :---: | :---: | :---: |
| (C,C) Low | 29 | 3 | 0 | 32 |
|  | $(2.64)$ | $(0.27)$ | $(0.00)$ | $(2.91)$ |
| (C,C) Average | 28 | 1019 | 0 | 1047 |
|  | $(2.55)$ | $(92.72)$ | $(0.00)$ | $(95.27)$ |
| (C,C) High | 0 | 16 | 4 | 20 |
|  | $(0.00)$ | $(1.46)$ | $(0.36)$ | $(1.82)$ |
| Total | 57 | 1038 | 4 | 1099 |
|  | $(5.19)$ | $(94.45)$ | $(.036)$ |  |

Table A.1: Hospital Cross Classifications of Mortality Rates by the (C,C) and (SLI,I) models.

