

Comparing the importance of disease rate versus practice style variations in explaining differences in small area hospitalization rates for two respiratory conditions

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SUMMARY

Many studies have reported large variations in age- and sex-adjusted rates of hospitalizations across small geographic areas. These variations have often been attributed to differences in medical practice style which are not reflected in differences in health care outcomes. There is, however, another potentially important source of variation that has not been examined much in the literature: geographic differences in the age–sex adjusted size of the pool of patients who present with the disease and are candidates for hospitalization. Previous studies of small area variations in hospitalization rates have only used data on hospitalizations. Thus, it has not been possible to distinguish the extent to which differences in hospitalization rates are due to (i) differences in the chance that patients diagnosed with a disease are admitted to a hospital, which we refer to as the ‘practice style effect,’ versus (ii) geographic differences in the total amount of diagnosed disease, which we refer to as the ‘disease effect.’ Elementary methods for estimating the relative strength of the two effects directly from the data can be misleading, since equal amounts of variability in each effect result in unequal impacts on hospitalization rates. In this paper we describe a model-based approach for estimating the relative importance of the practice style effect and the disease effect in explaining variations in hospitalization rates. The key to our approach is the use of data on both inpatient and outpatient visits. We use 1997 Medicare data for two respiratory medical conditions across 71 small areas in Massachusetts: chronic bronchitis and emphysema, and bacterial pneumonia. Based on a Poisson model for the process generating hospitalizations and outpatient visits, we use a Bayesian framework and Gibbs sampling to compute and compare the correlation between the number of people hospitalized and each of these two sources of variation. Our results show that for the two conditions, disease rate variation explains at least as much of the variation in hospitalization rates as does practice style variation. Copyright © 2003 John Wiley & Sons, Ltd.

KEY WORDS: small area variations; hospital utilization; practice style

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1. INTRODUCTION

Many studies have reported large variations in age- and sex-adjusted rates of hospitalizations across small geographical areas [1–13]. A widely held view, most clearly articulated by Wennberg and colleagues, is that ‘practice style’ differences account for much of the variation [14]. As stated concisely by Wennberg some years ago [15], small area studies have ‘conclusively demonstrated the following’: (i) ‘population characteristics such as age and morbidity cannot explain the degree of variation in health care utilization among small areas’; (ii) variation is ‘best explained by differences in clinical judgment on the appropriateness of treatment’; and (iii) ‘non-health factors such as supply of resources can influence clinical decisions’ (reference [15], p. 354). The implication sometimes drawn is that savings can be realized without sacrificing quality of care by reducing hospitalization rates in high rate areas to some benchmark defined by rates in the lower rate areas [16]. There is, however, another potentially important source of variation that has not been examined much in the literature: differences in the age- and sex-adjusted size of the pool of patients who present with the disease and are candidates for hospitalization. These differences could be explained by factors in addition to age and sex that result in differences in the amount of disease across areas [17, 18]. If these differences are important in explaining hospitalization rate variation, it shifts some of the focus of interventions from the clinical judgement of physicians to the identification of underlying risk factors giving rise to differences in disease rates and in initial access to the health care system.

Previous studies of small area variations in hospitalization rates have only used data on hospitalizations. Thus, it has not been possible to distinguish the extent to which differences in hospitalization rates are due to (i) differences in the likelihood that patients diagnosed with a disease are admitted to a hospital, which we refer to as the ‘practice style effect’, versus (ii) geographic differences in the total amount of diagnosed disease, which we refer to as differences in the amount of ‘identified’ disease or the ‘disease effect’. The key to distinguishing the magnitude of these effects is the use of data on both inpatient and outpatient visits. After adjusting areas for age and sex differences, we measure variation in the amount of ‘identified’ disease by looking at variation in the total number of people treated either as inpatients or outpatients. Practice style variation can be measured by looking at variation in the fraction of identified cases treated on an inpatient basis. The relative importance of each of these effects in explaining hospitalization rate variation is measured by calculating the correlation between two suitably chosen variables. It should be noted that the phrase ‘explaining variation’ implies only an association, in the same sense that in ordinary regression variation in an independent variable ‘explains’ variation in the dependent variable.

It is important to note that ‘identified’ disease is not the same thing as the underlying amount of disease. Since we ‘identify’ disease by diagnostic codes on patient bills, we are only able to measure that component of disease arising because a person sought and received care and because the physician coded the diagnosis. In a Medicare population that is Part B eligible (that is, participates in that part of the Medicare program that pays for outpatient care, which most of the elderly do), we believe that the major factor resulting in differences in the amount of ‘identified’ disease is differences in the amount of underlying disease. However, there may also be differences in initial access to physicians that influence the amount of ‘identified’ disease.

At first thought it might seem reasonable to compute a simple correlation coefficient between the number treated as inpatients and both (i) the total number treated as both inpatients and outpatients (which measures the disease effect) and (ii) the proportion of identified cases treated as inpatients (which measures the practice style effect). However, there are serious difficulties with this approach. Since both (i) and (ii) are increasing functions of the number of hospitalizations, a natural correlation will result. We show below how this leads to overestimates of the importance of each effect, and that the amount of overestimation differs for each effect.

Another seemingly reasonable approach might be to calculate the correlation between standardized inpatient and outpatient counts, and, if the correlation is positive, conclude that the 'disease effect' is stronger than the 'practice style effect'. This is motivated by the (easily justified) observation that under reasonable assumptions the correlation is non-negative when there is no practice style effect, and the correlation is non-positive when there is no disease effect. It unfortunately turns out that under these assumptions a positive correlation does not necessarily mean the disease effect is the stronger of the two effects. Hence, one must go beyond this elementary approach. Here we develop a statistical model for the process generating hospitalizations and then, using a Bayesian framework, estimate the relative strength of the two effects (variation in practice style and in amount of identified disease) in explaining variation in hospitalization rates. We use data from 1997 for 71 different geographical areas of Massachusetts for two medical conditions: chronic bronchitis and emphysema, and bacterial pneumonia.

The organization of the paper is as follows. In Section 2 we describe the construction of the data set used for analysis. In Section 3 we describe the statistical model and the use of Bayesian techniques to estimate model parameters. In addition, we report the results of two small simulations that illustrate that estimators of effects calculated directly from the data can be misleading, thus justifying the need for a model-based approach. In Section 4 we present estimates of the correlations between each effect and hospitalizations for the two conditions, the ratio of the correlations and variation of the two effects across small areas. We conclude in Section 5 with a discussion.

2. METHODS

2.1. Conditions

In an earlier study [19], we examined variation in hospitalization rates across small areas in Massachusetts for 68 'adjacent DRGs' (created by grouping DRGs previously split by age or complications/comorbidities), each of which had at least 1000 discharges in 1987 among persons 65 years and older and which consisted of a somewhat homogeneous group of diagnoses. For this study, we use data on two respiratory conditions: DRG 88: chronic bronchitis and emphysema (defined by ICD-9-CM codes 4911, 4912x, 4918, 4919, 4928, and 496); DRG 89/90: bacterial pneumonia (defined by ICD-9-CM codes 481, 4822, 4823x, 4829, 485 and 486). In our earlier study, DRG 88 was ranked 58th out of 68 in terms of variation across areas and DRG 89/90 42nd.

The physical symptoms and physiologic impact of both of these conditions are sufficiently uncomfortable and debilitating that people are likely to seek medical attention. With

COPD/emphysema, symptoms include shortness of breath, cough and reduced physical endurance; with bacterial pneumonia, they include fever, cough (often producing sputum or even blood), profound fatigue and weakness, pleuritic chest pain and shortness of breath. Some patients with pneumonia, most likely those in nursing homes who are not covered by Medicare, may die before seeking medical attention in a way captured by Medicare bills. Obviously, these patients would not be included in our analysis.

2.2. Identifying people who were hospitalized

Hospitalizations were identified from HCFA's 1997 MedPAR file, an analytic file created by HCFA based on inpatient bills. Because we did not have the Part B eligibility data, we only counted hospitalizations for those people who had at least one Part B bill in 1997 (thus ensuring their Part B eligibility for at least part of the year), slightly over 90 per cent of the population. For DRG 88, there were 4842 people hospitalized in 1997; for DRG 89/90, 8666 hospitalized.

2.3. Identifying people treated as outpatients only

The amount of identified disease is the sum of those treated as inpatients and those treated only on an outpatient basis. We identified outpatient visits from two 1997 Medicare files: the Carrier File (which has claims data for Part B physician/supplier services) and the Outpatient File (which has claims data for outpatient visits at hospitals and other institutions). A service was counted as an outpatient visit if it had an outpatient-related CPT code [20].

Outpatient diagnostic coding is less reliable and governed by somewhat different protocols than inpatient coding. To best capture the 'final' diagnosis associated with a string of outpatient visits, in the analysis reported here we used the following heuristic to associate ICD-9-CM codes with outpatient visits:

Any outpatient visit within 6 weeks of a previous outpatient visit was considered part of the same string of visits. The diagnostic codes on the last visit of a string were the codes assigned to that string of visits. An individual was eligible for inclusion in the count for a second 'string' only if there was at least an 8 week gap between the current visit and the end of the previous string.

If a diagnostic code associated with a string of visits was on the list of ICD-9-CM codes defining the DRGs of interest and if the patient was not hospitalized for the DRG, the patient was counted as having had outpatient-only treatment for that DRG. For DRG 88, there were 51 982 people treated on an outpatient-only basis, over 10 times the number treated as inpatients; for DRG 89/90, there were 7899 people treated on an outpatient-only basis, slightly less than the number treated as inpatients. Thus, in terms of likelihood of hospitalization, these are two very different conditions.

We examined the sensitivity of our conclusions to several alternative ways of counting outpatient-only treatment for each DRG. First, rather than only considering diagnoses at the end of a string of visits, we used the diagnostic codes at each outpatient visit as the basis for placing patients in a DRG. Second, for both the string method and when strings were ignored and each visit considered separately, we just used the first listed diagnosis on each bill as the basis for placing a patient in a DRG. Though the number of people receiving outpatient-only

treatment was different for each of the methods, our substantive conclusions were insensitive to the method of determining outpatient-only counts [20].

2.4. Creating small geographic areas

Small geographic areas were formed using Ward's clustering algorithm [21, 22], to group zip codes displaying a similar pattern of hospital discharges, that is, zip codes in which the proportion of overall discharges that were from each hospital used by zip code residents were similar. Discharges of patients with the following characteristics were included in the clustering: the patient was 65 years or older and resided in Massachusetts; and the discharge was in 1997 and was from a hospital in Massachusetts that was paid under the Prospective Payment System. Based on results in our earlier study [19], we stopped the clustering at 70 small areas. Several *ad hoc* adjustments were then made to ensure small areas consisted of geographically contiguous zip codes and to eliminate several very small areas [20]. After these adjustments, there were 71 small areas.

2.5. The data set for analysis

For each of the two DRGs, the analytical data set consisted of the following for each of the 71 areas: the observed number of people hospitalized; the observed number of people treated on an outpatient-only basis; the expected number of people hospitalized given the age–sex distribution in the area and statewide age–sex hospitalization rates, and the expected number treated on an outpatient-only basis. Expected numbers were calculated using indirect standardization.

3. THE STATISTICAL MODEL

Our approach, as described in this section, is to (i) develop a model to explain the data, (ii) use the data to estimate model parameters, and (iii) use the model parameters to shed light on the relative importance of the different sources of variation or effects.

Consider one of the conditions. For each of the 71 areas, the data consisted of the following four numbers:

O_i = observed number of people hospitalized in the area (that is, treated as inpatients)

O_o = observed number of treated on an outpatient-only basis in the area

and

E_i and E_o , the corresponding expected numbers computed by indirect standardization

Our model for these data contains a total of four unknown parameters $\gamma_1, \gamma_2, \gamma_3, \gamma_4$ which we next describe.

We construct a model by first assuming that the total pool $O_i + O_o$ of identified disease follows a Poisson distribution with a mean which is itself a random variable. This mean depends on the expected size of the identified disease pool $E_i + E_o$ along with an independent factor α which represents variation in the overall expected amount of identified disease. Specifically, we suppose that in each of the 71 areas

$$(O_i + O_o) | \alpha \sim \text{Poisson}(\alpha(E_i + E_o))$$

where α is a random variable independent from area to area, which for simplicity we assume to have a common gamma distribution

$$\alpha \sim \text{Gamma}(\gamma_1, \gamma_2)$$

Here α is a random scale factor representing geographical variation in the size of the total pool of identified disease. The assumption of a gamma distribution is a mild assumption, made primarily to ensure $\alpha > 0$.

Next we suppose that within each area the allocation to the two different treatment options (inpatient and outpatient-only) occurs randomly and independently according to a probability which is itself variable. Variation in this probability represents practice style variation. Specifically, we suppose that

$$(O_i | O_i + O_o, \beta) \sim \text{Binomial} \left(O_i + O_o, \beta \frac{E_i}{E_i + E_o} \right)$$

where

$$\beta \sim \text{Gamma}(\gamma_3, \gamma_4)$$

is a random variable independent and identically distributed from area to area. (For technical and computational purposes we restrict the binomial probability to be less than 1 to guard against the very rare chance of having an invalid probability. This would crash the software used to estimate the model parameters, and is a very mild modification. We prefer this to the use of a logit function in order to simplify estimation of correlations later.) The above model indicates that, given the size of the identified disease pool, each case is treated on an inpatient basis independently with probability $\beta \frac{E_i}{E_i + E_o}$, so that β serves to scale up or down the expected probability by some factor. β is a random variable representing geographical variation in practice style. We further assume that within an area α and β are independent of each other – an assumption that is supported by the data (see Appendix B).

To examine the relative importance of practice style versus amount of identified disease in explaining variations in hospitalization rates, we compare $\text{corr}(\alpha, O_i)$ and $\text{corr}(\beta, O_i)$. As mentioned before, the phrase ‘explaining variation’ implies only an association, not causality.

The next proposition shows that under the model these correlations are completely determined by of the first two moments of α and β , which can be expressed in terms of the unknown parameters $\gamma_1 \dots \gamma_4$. We treat E_i and E_o as constants, so the entire calculation we make is made conditional on these:

Proposition 1

Under the above model

$$\begin{aligned} \text{corr}(\alpha, O_i) &= E[\beta] \text{SD}(\alpha) / \sqrt{\{E[\beta^2]E[\alpha^2] - (E[\beta]E[\alpha])^2 + E[\beta]E[\alpha]/E_i\}} \\ &= \sqrt{\{\gamma_3 / (1 + \gamma_3 + \gamma_1 + \gamma_2\gamma_4/E_i)\}} \end{aligned}$$

and

$$\begin{aligned} \text{corr}(\beta, O_i) &= E[\alpha] \text{SD}(\beta) / \sqrt{\{E[\beta^2]E[\alpha^2] - (E[\beta]E[\alpha])^2 + E[\beta]E[\alpha]/E_i\}} \\ &= \sqrt{\{\gamma_1 / (1 + \gamma_3 + \gamma_1 + \gamma_2\gamma_4/E_i)\}} \end{aligned}$$

Proof

See Appendix A.

It immediately follows that

$$\frac{\text{corr}(\alpha, O_i)}{\text{corr}(\beta, O_i)} = \frac{E[\beta]\text{SD}(\alpha)}{E[\alpha]\text{SD}(\beta)} = \frac{cv(\alpha)}{cv(\beta)} = \sqrt{(\gamma_3/\gamma_1)}$$

indicating that the factor with the higher coefficient of variation has a stronger correlation with observed hospitalizations.

Thus, the problem of estimating the correlations reduces to the problem of estimating the parameters $\gamma_1 \dots \gamma_4$. For this we use a Bayesian approach, assuming diffuse prior distributions for γ_i and then computing the posterior distribution of the correlations and their ratio given the data. We use the prior distributions

$$\gamma_i \sim \text{uniform}(0, 1000)$$

and use the Gibbs sampling software package WINBUGS to compute the posterior distributions of the correlations (using the above proposition) conditional on the data based on 5000 samples each, after a 'warm-up' period of 300 000 samples.

It is interesting to note that α and β are hidden variables, and they can only be estimated from the data. At first thought it might seem reasonable to use the estimates

$$\alpha_{\text{est}} = \frac{O_i + O_o}{E_i + E_o}$$

and

$$\beta_{\text{est}} = \frac{O_i}{O_i + O_o} \bigg/ \frac{E_i}{E_i + E_o}$$

and then use $\text{corr}(\alpha_{\text{est}}, O_i)$ and $\text{corr}(\beta_{\text{est}}, O_i)$ as estimates of $\text{corr}(\alpha, O_i)$ and $\text{corr}(\beta, O_i)$. This will not work well because, as noted earlier, both α_{est} and β_{est} are increasing functions of O_i . It is easy to see that this would result in overestimates of the two correlations, and that each could overestimate by a different amount. As an example suppose O_i and O_o are independent Poisson random variables with means $E_i = 25$ and $E_o = 100$, respectively. A straightforward simulation shows that $\text{corr}(\alpha_{\text{est}}, O_i) \approx 0.4$ and $\text{corr}(\beta_{\text{est}}, O_i) \approx 0.9$. It is also the case that even the relative spread of α_{est} and β_{est} can be misleading. For example, under the assumption that both α and β have the same distribution (gamma with both parameters = 40) and again $E_i = 25$ and $E_o = 100$, a simulation shows that the standard deviation of α_{est} is approximately 0.18 and for β_{est} is approximately 0.24.

4. RESULTS

The correlations $\text{corr}(\alpha, O_i)$ and $\text{corr}(\beta, O_i)$ depend on E_i , the expected number of inpatient visits. Thus for each medical condition there are different estimates of the correlation for each of the 71 areas. In Table I are estimates of the correlations for the smallest, median and largest areas for each of the two conditions. As can be seen, for all areas and conditions, the estimated disease effect is slightly stronger than the practice style effect.

Table I. Correlation of number of hospitalizations with the disease effect and the practice style effect for the largest, median and smallest areas, by DRG.

	Correlation with number of hospitalizations	
	DRG 88	DRG 89/90
<i>Largest area</i>		
Disease effect	0.73	0.70
Practice style effect	0.63	0.67
<i>Median area</i>		
Disease effect	0.70	0.65
Practice style effect	0.60	0.63
<i>Smallest area</i>		
Disease effect	0.43	0.37
Practice style effect	0.37	0.36

Though the correlation of each effect depends on the size of the area, the ratio of the correlations does not. In what follows, we list for each condition the ratio of $\text{corr}(\alpha, O_i)$ to $\text{corr}(\beta, O_i)$ and the 95 per cent Bayesian prediction intervals:

$$\begin{aligned} \text{DRG 88: } & 1.15(0.87, 1.55) \\ \text{DRG 89/90: } & 1.05(0.75, 1.46) \end{aligned}$$

Consistent with the correlations reported in Table I, the disease effect is stronger than the practice style effect. However, the difference is not statistically significant.

Both the disease effect and the practice style effect are centred around a number close to 1. For DRG 88, the standard deviation of the disease effect is 0.225; the standard deviation of the practice style effect is 0.197. For DRG 89/90, the standard deviation of the disease effect is 0.143; the standard deviation of the practice style effect is 0.134. Thus, in both cases, the disease effect is slightly more variable across areas than the practice style effect.

As a check of the numerical techniques, we generated random values for the observed counts under the assumption that $\alpha = \beta = 1$ and estimated the correlations for this simulated data. In this case, the correlations are expected to be zero. In fact, in every case the estimated correlation is below 0.10.

5. DISCUSSION

Our model is an extension of the model initially proposed by McPherson *et al.* [8] to estimate the systematic component of variation. They assumed that the observed number of hospitalizations in an area follows a Poisson distribution with mean λE_i , where E_i is the expected number of hospitalizations in the area calculated using indirect standardization and λ is an area-specific factor reflecting the extent to which the area differs from expected. The variance of the λ 's across areas, σ^2 , is an estimate of the systematic coefficient of variation. McPherson *et al.* estimate this as the average of area-specific estimates of systematic variation, which can be calculated from the underlying model. In our earlier work [19], using an empirical Bayes

framework, an alternative estimate for σ^2 was derived, one that is a weighted average of the area-specific estimates, where the weights are a function of the size of the areas.

We have extended this initial model to the situation in which one considers both the number of people hospitalized and number treated on an outpatient-only basis. In our case, the observed number of people hospitalized in an area is essentially assumed to follow a Poisson distribution with mean $\alpha\beta E_i$ (see Appendix A), where α indicates the disease effect and β the practice style effect. Using a Bayesian framework and Gibbs sampling, we simulated the distribution of the α 's and β 's and estimated their standard deviation, a measure comparable to the systematic component of variation. In addition, we have shown that the relative strength of the association between number hospitalized and the disease effect versus the practice style effect depends on the ratio of the coefficient of variation of these two effects.

It is important to note that whereas most previous studies of small area variations in hospitalizations focus on variation in number of hospitalizations, we consider variation in the number of people hospitalized. Most of the variation in hospitalizations is caused by variation in number of people hospitalized [20]. The main reason we consider number of people rather than number of visits has to do with outpatient visits. Whereas the initial outpatient visit is most driven by illness (for example, Wennberg notes [15] 'small area studies are also compatible with the notion that illness is the major factor in the individual patient's decision to consult a physician'), the number of outpatient visits may well be influenced by differences in practice style, which in turn may be influenced by per capita physician supply. Also, the Poisson distribution is a more reasonable assumption for counts of number of people than for total utilization. Readmissions and revisits are likely to increase variation over that implied by a Poisson distribution.

Our important substantive conclusion from this analysis is that for the two respiratory conditions we examined, disease rate variation explains at least as much variation in the observed number hospitalized as does practice style variation. This implies that for these conditions, age and sex standardization do not adequately account for differences in the amount of the disease across areas, an argument proposed some years ago by Blumberg [17]. In response, Wennberg makes the argument that 'population illness rates do not explain population hospitalization rates' [15]. Based on surveys of the population in different hospital market areas across Vermont, Wennberg and Fowler found little differences in morbidity across areas [23]. Our contribution to this argument has been to analyse data on outpatient visits, which allows us to distinguish the relative strength of the association of hospitalizations with amount of disease versus practice style. This analysis clearly suggests, at least for the conditions we looked at, the importance of variation in amount of disease across areas.

APPENDIX A: PROOF OF PROPOSITION 1

First note that since an independent binomial splitting of a Poisson process yields a Poisson process, it can be easily seen that

$$O_i|\alpha, \beta \sim \text{Poisson}(\alpha\beta E_i)$$

Next we have

$$\text{corr}(\alpha, O_i) = \frac{E[\alpha O_i] - E[\alpha]E[O_i]}{\text{SD}(\alpha)\text{SD}(O_i)}$$

where we assume here and in what follows that all expectations, standard deviations and variances are calculated with respect to only the variables O_i, α, β (viewing the parameters for these variables $\gamma_1 \dots \gamma_4$ as fixed). We use the first representation above along with the stated assumption to get

$$E[\alpha O_i] = E[E[\alpha O_i | \alpha, \beta]] = E[\alpha^2 \beta E_i] = E[\alpha^2] E[\beta] E_i$$

and

$$E[\alpha] E[O_i] = E[\alpha] E[E[O_i | \alpha, \beta]] = E[\alpha] E[\alpha \beta E_i] = (E[\alpha])^2 E[\beta] E_i$$

Furthermore

$$\text{var}(O_i) = E[\text{var}(O_i | \alpha, \beta)] + \text{var}(E[O_i | \alpha, \beta])$$

where

$$E[\text{var}(O_i | \alpha, \beta)] = E[\alpha] E[\beta] E_i$$

and

$$\text{var}(E[O_i | \alpha, \beta]) = \text{var}(\alpha \beta E_i) = (E_i)^2 (E[\alpha^2] E[\beta^2] - (E[\alpha] E[\beta])^2)$$

Putting together the pieces and simplifying gives

$$\text{corr}(\alpha, O_i) = E[\beta] \text{SD}(\alpha) / \sqrt{\{E[\beta^2] E[\alpha^2] - (E[\beta] E[\alpha])^2 + E[\beta] E[\alpha] / E_i\}}$$

Since with the gamma distribution $E[\alpha] = \gamma_1 / \gamma_2$ and $\text{var}(\alpha) = \gamma_1 / (\gamma_2)^2$, we have

$$\begin{aligned} \text{corr}(\alpha, O_i) &= \frac{\gamma_3 \gamma_1}{\gamma_2 \gamma_4} \bigg/ \sqrt{\left\{ \frac{\gamma_1(\gamma_1 + 1)}{(\gamma_2)^2} \frac{\gamma_3(\gamma_3 + 1)}{(\gamma_4)^2} - \left(\frac{\gamma_1}{\gamma_2} \frac{\gamma_3}{\gamma_4} \right)^2 + \frac{\gamma_1}{\gamma_2} \frac{\gamma_3}{\gamma_4} \bigg/ E_i \right\}} \\ &= \gamma_3 \gamma_1 / \sqrt{\{\gamma_1 \gamma_3^2 + \gamma_1 \gamma_3 + \gamma_3 \gamma_1^2 + \gamma_1 \gamma_2 \gamma_3 \gamma_4 / E_i\}} \\ &= \sqrt{\{\gamma_3 / (1 + \gamma_3 + \gamma_1 + \gamma_2 \gamma_4 / E_i)\}} \end{aligned}$$

the formula given for $\text{corr}(\alpha, O_i)$. The formula for $\text{corr}(\beta, O_i)$ follows by symmetry.

APPENDIX B: REASONABLENESS OF THE ASSUMPTION THAT THE PRACTICE STYLE EFFECT AND THE DISEASE EFFECT ARE INDEPENDENT

We investigated this in two ways. For each DRG, we first calculated the correlation between (heuristically) standardized deviates for each effect, that is

$$\text{corr} \left(\frac{O_i + O_o - (E_i + E_o)}{E_i + E_o}, \frac{(O_i / O_o - E_i / E_o)}{\sqrt{\{E_i / (E_i + E_o) (1 - E_i / (E_i + E_o)) / (E_i + E_o)\}}} \right)$$

For both DRG 88 and DRG 89/90, the correlation was 0.03 and the scatter plot showed no visible signs of dependence. This supports the assumption of independence.

Secondly, we modified our model to allow for the possibility that α_j and β_j are linked by supposing that

$$\beta_j = (1 + k\alpha_j)\varepsilon_j$$

where k is some unknown constant and ε_j is a random variable independent of all else. We assumed ε_j followed a gamma distribution with unknown parameters (with non-informative uniform 0 to 1000 priors on the parameters), and used a random variable uniform over the range -1 to $+1$ as a prior for k . After fitting the model via Gibbs sampling, we found that for either DRG k was not significantly different from zero, in the sense that the 95 per cent Bayesian prediction intervals covered zero. From these two approaches we concluded that, at least for the two conditions we examined, the assumption of the independence of α_j and β_j is reasonable.

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