

Introduction to Bayesian Data Analysis

4: Bayesian Random-Effects Hierarchical Modeling

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4.1 Formulating hierarchical models for quantitative outcomes from scientific context

Meta-analysis of effects of aspirin on heart attacks. Table 2.1 (Draper et al., 1993a) gives the number of patients and mortality rate from all causes, for six randomized controlled experiments comparing the use of aspirin and placebo by patients following a heart attack.

Table 2.1. Aspirin meta-analysis data.

Study (i)	Aspirin		Placebo	
	# of Patients	Mortality Rate (%)	# of Patients	Mortality Rate (%)
UK-1	615	7.97	624	10.74
CDPA	758	5.80	771	8.30
GAMS	317	8.52	309	10.36
UK-2	832	12.26	850	14.82
PARIS	810	10.49	406	12.81
AMIS	2267	10.85	2257	9.70
Total	5599	9.88	5217	10.73

Study (i)	Comparison		Z_i^\ddagger	p_i^\S
	$y_i = \text{Diff} (\%)$	$\sqrt{V_i} = \text{SE of Diff} (\%)$		
UK-1	2.77	1.65	1.68	.047
CDPA	2.50	1.31	1.91	.028
GAMS	1.84	2.34	0.79	.216
UK-2	2.56	1.67	1.54	.062
PARIS	2.31	1.98	1.17	.129
AMIS	-1.15	0.90	-1.27	.898
Total	0.86	0.59	1.47	.072

$^\ddagger Z_i$ denotes the ratio of the difference in mortality rates over its standard error, assuming a binomial distribution. $^\S p_i$ is the one-sided p value associated with Z_i , using the normal approximation.

Meta-Analysis

The first five trials are reasonably consistent in showing a (weighted average) mortality decline for aspirin patients of 2.3 percentage points, a 20% drop from the (weighted average) placebo mortality of 11.5% (this difference is **highly clinically significant**). However, the sixth and largest trial, AMIS, went the other way: an *increase* of 1.2 percentage points in aspirin mortality (a 12% rise from the placebo baseline of 9.7%).

Some relevant questions (Draper, 1995): Q₁ Why did AMIS get such different results? Q₂ What should be done next to reduce the uncertainty about Q₁? Q₃ If I were a doctor treating a patient like those eligible for the trials in Table 2.1, what therapy should I employ while answers to Q₁ and Q₂ are sought? One possible paraphrase of Q₃: Q₄ How should the information from these six experiments be **combined** to produce a more informative summary than those obtained from each experiment by itself?

The discipline of meta-analysis is devoted to answering questions like Q₄. One leading school of frequentist meta-analysis (e.g., Hedges and Olkin, 1985) looks for methods for combining the Z and p values in Table 2.1, an approach that often leads only to an overall p value. A more satisfying form of meta-analysis (which has both frequentist and Bayesian versions) builds a **hierarchical model (HM)** that indicates how to combine information from the mortality differences in the Table. A Gaussian meta-analysis model for the aspirin data, for example (Draper et al., 1993a), might look like

$$\begin{array}{llll}
 (\theta, \sigma^2) & \sim & p(\theta, \sigma^2) & \text{(prior)} \\
 (\theta_i | \theta, \sigma^2) & \stackrel{\text{IID}}{\sim} & N(\theta, \sigma^2) & \text{(underlying effects)} \\
 (y_i | \theta_i) & \stackrel{\text{indep}}{\sim} & N(\theta_i, V_i) & \text{(data)} .
 \end{array} \tag{1}$$

A Gaussian HM

The bottom level of (10), the **data** level of the HM, says that—because of relevant differences in patient cohorts and treatment protocols—each study has its own underlying treatment effect θ_i , and the observed mortality differences y_i are like random draws from a normal distribution with mean θ_i and variance V_i (the normality is reasonable because of the CLT, given the large numbers of patients). In meta-analyses of data like those in Table 2.1 the V_i are typically taken to be known (again because the patient sample sizes are so big), $V_i = SE_i^2$, where SE_i is the standard error of the mortality difference for study i in Table 2.1.

The middle level of the HM is where I would bring in the **study-level covariates**, if I have any, to try to explain why the studies differ in their underlying effects. Here there are no study-level covariates, so the middle level of (10) is equivalent to a Gaussian regression with no predictor variables. Why the “error” distribution should be Gaussian at this level of the HM is not clear—it’s a conventional choice, not a choice that is automatically scientifically reasonable (in fact I will challenge it later). σ^2 in this model represents **study-level heterogeneity**.

The top level of (10) is where the **prior** distribution on the regression parameters from the middle level is specified. Here, with only an intercept term in the regression model, a popular conventional choice is a normal/**scaled-inverse- χ^2** prior (more about this later; see Gelman et al. 2003).

Fixed effects and random effects. If σ^2 were known somehow to be 0, all of the θ_i would have to be equal deterministically to a common value θ , yielding a simpler model: $(y_i|\theta) \stackrel{\text{indep}}{\sim} N(\theta, V_i), \theta \sim p(\theta)$. Meta-analysts call this a **fixed-effects** model, and refer to model (10) as a **random-effects** model. When σ^2 is not assumed to be 0, with this terminology the θ_i are called **random effects**.

4.2 Approximate Fitting of Gaussian Hierarchical Models: Maximum Likelihood and Empirical Bayes

Fitting HM (10). Some algebra based on model (10) yields that the conditional distributions of the study-level effects θ_i given the data and the parameters (θ, σ^2) , have a simple and revealing form:

$$(\theta_i | y_i, \theta, \sigma^2) \stackrel{\text{indep}}{\sim} N(\theta_i^*, V_i(1 - B_i)) \quad (2)$$

$$\text{with } \theta_i^* = (1 - B_i) y_i + B_i \theta \quad \text{and} \quad B_i = \frac{V_i}{V_i + \sigma^2}. \quad (3)$$

In other words, the conditional mean of the effect for study i given y_i, θ , and σ^2 is a **weighted average** of the sample mean for that study, y_i , and the overall mean θ .

The weights are given by the so-called **shrinkage factors** B_i (e.g., Draper et al., 1993a), which in turn depend on how the variability V_i within study i compares to the between-study variability σ^2 : the more accurately y_i estimates θ_i , the more weight the “local” estimate y_i gets in the weighted average.

The term *shrinkage* refers to the fact that, with this approach, unusually high or low individual studies are drawn back or “shrunk” toward the overall mean in the calculation $(1 - B_i) y_i + B_i \theta$. Note that θ_i^* uses data from all the studies to estimate the effect for study i —this is referred to as **borrowing strength** in the estimation process.

Closed-form expressions for $p(\theta | y)$ and $p(\theta_i | y)$ with $y = (y_1, \dots, y_k)$, $k = 6$ are not available even with a normal/scaled-inverse- χ^2 prior for (θ, σ^2) ; a full Bayesian analysis of these data is most readily accomplished with **Markov chain Monte Carlo** methods, to which we will soon turn.

Maximum Likelihood and Empirical Bayes

In the meantime **maximum likelihood** calculations provide some idea of what to expect: the likelihood function based on model (10) is

$$l(\theta, \sigma^2 | y) = c \prod_{i=1}^k \frac{1}{\sqrt{V_i + \sigma^2}} \exp \left[-\frac{1}{2} \sum_{i=1}^k \frac{(y_i - \theta)^2}{V_i + \sigma^2} \right]. \quad (4)$$

The maximum likelihood estimates (MLEs) $(\hat{\theta}, \hat{\sigma}^2)$ then turn out to be the **iterative** solutions to the following equations:

$$\hat{\theta} = \frac{\sum_{i=1}^k \hat{W}_i y_i}{\sum_{i=1}^k \hat{W}_i} \quad \text{and} \quad \hat{\sigma}^2 = \frac{\sum_{i=1}^k \hat{W}_i^2 [(y_i - \hat{\theta})^2 - V_i]}{\sum_{i=1}^k \hat{W}_i^2}, \quad (5)$$

$$\text{where} \quad \hat{W}_i = \frac{1}{V_i + \hat{\sigma}^2}. \quad (6)$$

Start with $\hat{\sigma}^2 = 0$ and iterate (14–15) to convergence (if $\hat{\sigma}^2$ converges to a negative value, $\hat{\sigma}^2 = 0$ is the MLE). The MLEs of the θ_i are then given by

$$\hat{\theta}_i = (1 - \hat{B}_i) y_i + \hat{B}_i \theta \quad \text{where} \quad \hat{B}_i = \frac{V_i}{V_i + \hat{\sigma}^2}. \quad (7)$$

These are called **empirical Bayes** (EB) estimates of the study-level effects, because it turns out that this analysis approximates a fully Bayesian solution by (in effect) using the data to estimate the prior specifications for θ and σ^2 .

Large-sample (mainly meaning large k) approximations to the (frequentist) distributions of the MLEs are given by

$$\hat{\theta} \sim N \left(\theta, \left[\sum_{i=1}^k \frac{1}{V_i + \hat{\sigma}^2} \right]^{-1} \right) \quad \text{and} \quad \hat{\theta}_i \sim N(\theta_i, V_i(1 - \hat{B}_i)). \quad (8)$$

MLEB (continued)

NB The variances in (17) *do not account fully for the uncertainty in σ^2* and therefore underestimate the actual sampling variances for small k (adjustments are available; see, e.g., Morris (1983, 1988)).

MLEB estimation can be implemented simply in about 15 lines of S+ or R code (Table 2.2).

Table 2.2. S+ or R program to perform MLEB calculations.

```

mleb <- function( y, V, m ) {
  sigma.2 <- 0.0
  for ( i in 1:m ) {
    W <- 1.0 / ( V + sigma.2 )
    theta <- sum( W * y ) / sum( W )
    sigma.2 <- sum( W^2 * ( ( y - theta )^2 - V ) ) / sum( W^2 )
    B <- V / ( V + sigma.2 )
    effects <- ( 1 - B ) * y + B * theta
    se.theta <- 1.0 / sqrt( sum( 1.0 / ( V + sigma.2 ) ) )
    se.effects <- sqrt( V * ( 1.0 - B ) )
    print( c( i, theta, se.theta, sigma.2 ) )
    print( cbind( W, ( W / sum( W ) ), B, y, effects, se.effects ) )
  }
}

```

With the aspirin data it takes 18 iterations (less than 0.1 second on a 400MHz UltraSPARC Unix machine) to get 4-digit convergence to the summaries in Table 2.3 and the following estimates (standard errors in parentheses):

$$\hat{\theta} = 1.45 (0.809), \quad \hat{\sigma} = 1.24.$$

Table 2.3. Maximum likelihood empirical Bayes meta-analysis of the aspirin data.

study(i)	\hat{W}_i	normalized \hat{W}_i	\hat{B}_i	y_i	$\hat{\theta}_i$	$\widehat{SE}(\hat{\theta}_i)$
1	0.235	0.154	0.640	2.77	1.92	0.990
2	0.308	0.202	0.529	2.50	1.94	0.899
3	0.143	0.0934	0.782	1.84	1.53	1.09
4	0.232	0.151	0.646	2.56	1.84	0.994
5	0.183	0.120	0.719	2.31	1.69	1.05
6	0.427	0.280	0.346	-1.15	-0.252	0.728

Aspirin Meta-Analysis: Conclusions

Note that (1) AMIS gets much less weight (normalized \widehat{W}_i) than would have been expected given its small V_i ; (2) the shrinkage factors (\widehat{B}_i) are considerable, with AMIS shrunk almost all the way into positive territory (see Figure 2.1); (3) there is **considerable study-level heterogeneity** ($\widehat{\sigma} \doteq 1.24$ percentage points of mortality); and (4) the standard errors of the effects are by and large smaller than the $\sqrt{V_i}$ (from the borrowing of strength) but are still considerable.

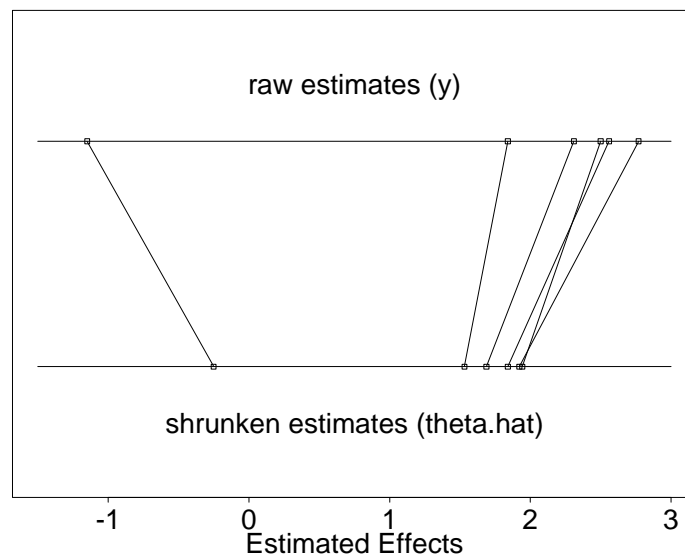


Figure 2.1. **Shrinkage plot** for the aspirin MLEB meta-analysis.

The 95% interval estimate of θ , the overall underlying effect of aspirin on mortality, from this approach comes out

$$\widehat{\theta} \pm 1.96 \cdot \widehat{SE}(\widehat{\theta}) \doteq (-0.140, 3.03),$$

which if interpreted Bayesianly gives

$$P(\text{aspirin reduces mortality}|\text{data}) \doteq 1 - \Phi\left(\frac{0-1.45}{0.809}\right) = \mathbf{0.96},$$

where Φ is the standard normal CDF. Thus although the interval includes 0, so that in a frequentist sense the effect is not statistically significant, **in fact from a Bayesian point of view the evidence is running strongly in favor of aspirin's usefulness.**

WinBUGS Analysis of Aspirin Data

Aspirin meta-analysis revisited. I create three files for WinBUGS: a **model** file, a **data** file, and an **initial values** file (I'm using the most recent release, 1.4.1, of WinBUGS).

The (first) **model** file for the aspirin data:

```
{  
  
mu ~ dnorm( 0.0, 1.0E-6 )  
tau.theta ~ dgamma( 1.0E-3, 1.0E-3 )  
  
for ( i in 1:k ) {  
  
    theta[ i ] ~ dnorm( mu, tau.theta )  
    y[ i ] ~ dnorm( theta[ i ], tau.y[ i ] )  
  
}  
  
sigma.theta <- 1.0 / sqrt( tau.theta )  
  
}
```

WinBUGS Analysis of Aspirin Data

Here μ plays the role of θ in model (10) above to avoid using the name `theta` twice for two different purposes in the WinBUGS program.

In specifying a normal distribution WinBUGS works not with a **standard deviation** (SD) or a **variance** but with a **precision**—the **reciprocal** of the variance—so that the $N(\mu, \sigma^2)$ distribution is specified by `dnorm(mu, tau)` with $\tau = \frac{1}{\sigma^2}$.

Then the **SD** has to be computed as a derived quantity ($\sigma = \frac{1}{\sqrt{\tau}}$) which is written above as
`sigma.theta <- 1.0 / sqrt(tau.theta)`

If—before the aspirin experiments were performed—I'm relatively **ignorant** about the quantities θ (μ) and σ in model (10), or equivalently μ and $\tau = \frac{1}{\sigma^2}$, I can build a **diffuse** or **flat** prior for both quantities that expresses this relative ignorance.

Since μ lives on $(-\infty, \infty)$ a convenient choice for a flat prior for it is a **normal** distribution with mean (say) 0 and very small precision: `mu ~ dnorm(0.0, 1.0E-6)`

For `tau.theta`, which lives on $(0, \infty)$, I want something that's flat throughout (almost) all of that range; a convenient choice (to get an **initial idea** of where the posterior distribution for `sigma.theta` is **concentrated**) is a **gamma** distribution with small positive values of both of its parameters.

This is the $\Gamma(\epsilon, \epsilon)$ distribution for some **small** $\epsilon > 0$ like 0.001: `tau.theta ~ dgamma(1.0E-3, 1.0E-3)`

WinBUGS Aspirin Analysis (continued)

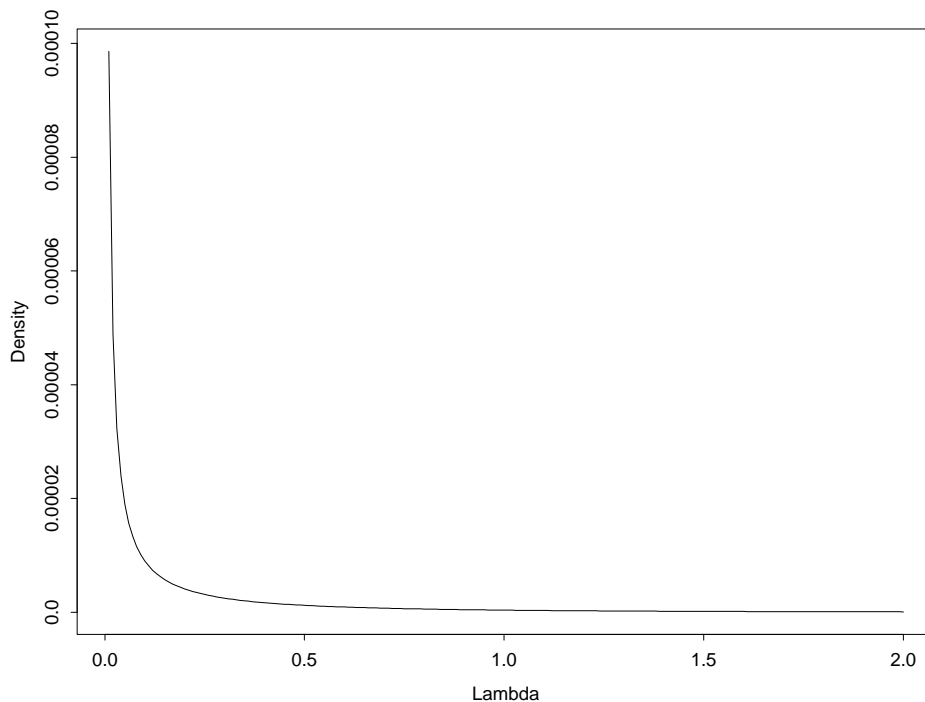


Figure 3.1. The $\Gamma(0.001, 0.001)$ distribution.

The **data** file in the aspirin meta-analysis is

```
list( k = 6, y = c( 2.77, 2.50, 1.84, 2.56, 2.31, -1.15 ),  
      tau.y = c( 0.3673, 0.5827, 0.1826, 0.3586, 0.2551, 1.235 ) )
```

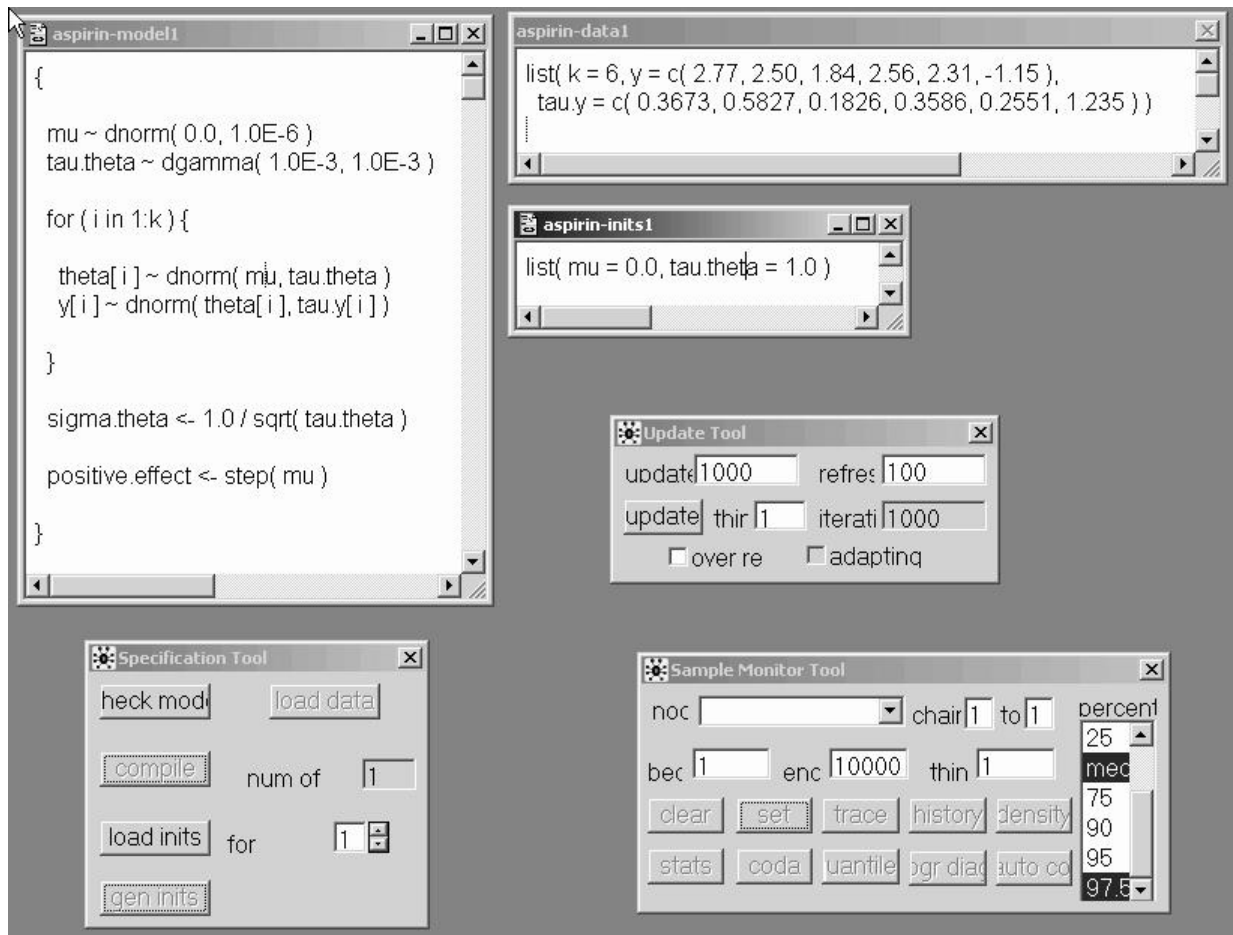
Here, e.g., $\text{tau.y}[1] = \frac{1}{1.65^2} \doteq 0.3673$, where 1.65 is the **standard error** of the difference $y[1]$ for experiment 1 in Table 2.1 on p. 20.

Finally, the **initial values** file in the aspirin meta-analysis is

```
list( mu = 0.0, tau.theta = 1.0 )
```

In a simple example like this there's no harm in starting the Markov chain off in a **generic** location: since μ and τ_θ live on $(-\infty, \infty)$ and $(0, \infty)$, convenient generic choices for their starting values are 0 and 1, respectively (more care may be required in models with **more complex random-effects structure**).

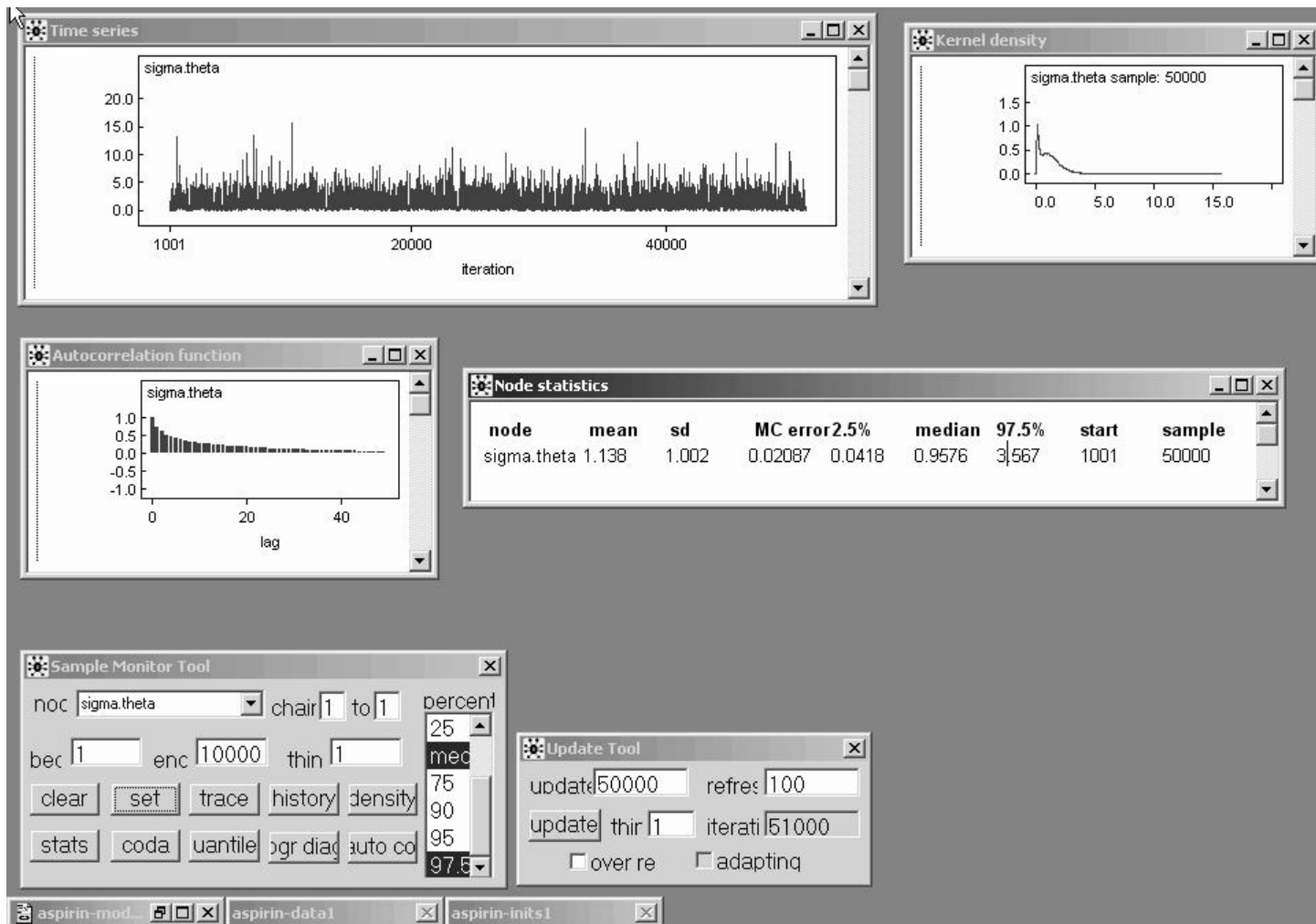
WinBUGS Aspirin Analysis (continued)



I (1) get a Specification Tool from the Model menu, (2) click on the **model** window and click check model, (3) click on the **data** window and click load data and compile, (4) click on the **initial values** window and click load inits, and (5) click gen inits (because the random effects θ_i were uninitialized in the inits file); I'm now ready to do some MCMC sampling.

I (6) get an Update Tool from the Model menu, and click update to perform a **burn-in** of 1,000 iterations (the default), which takes **0s** at 1.6 Pentium GHz; (7) I then get a Sample Monitoring Tool from the Inference menu, and type `sigma.theta` and click set.

WinBUGS Aspirin Analysis (continued)



(8) I type 50000 in the updates window in the Update Tool and click update to get a **monitoring** run of **50,000** iterations (this took **15s**).

Then (9) selecting sigma.theta in the node window, all 10 buttons from clear through autoC are active, and I click on history (to get a Time Series window), density (to get a Kernel density window), autoC to get an Autocorrelation function window, and stats (to get a Node statistics window), **yielding the screen above**.

The output of an MCMC sampler, when considered as a **time series**, often exhibits **positive autocorrelation**; in fact it often looks like a realization of an **autoregressive** AR_p model of order $p = 1$ ($\theta_t = \alpha + \beta\theta_{t-1} + e_t$) with **positive first-order autocorrelation** ρ .

WinBUGS Aspirin Analysis (continued)

This does not affect the **validity** of Monte Carlo inferences about the unknowns (e.g., the mean of any **stationary stochastic process** is a **consistent** estimator of the underlying process mean), but it does affect the **efficiency** of these inferences: for example, the Monte Carlo variance of the sample mean $\bar{\theta}$ based on M draws from an AR_1 time series is

$$V(\bar{\theta}) = \frac{\sigma_{\theta}^2}{M} \left(\frac{1 + \rho}{1 - \rho} \right), \quad (9)$$

and the **sample size inflation factor** $\frac{1+\rho}{1-\rho} \rightarrow \infty$ as $\rho \rightarrow +1$.

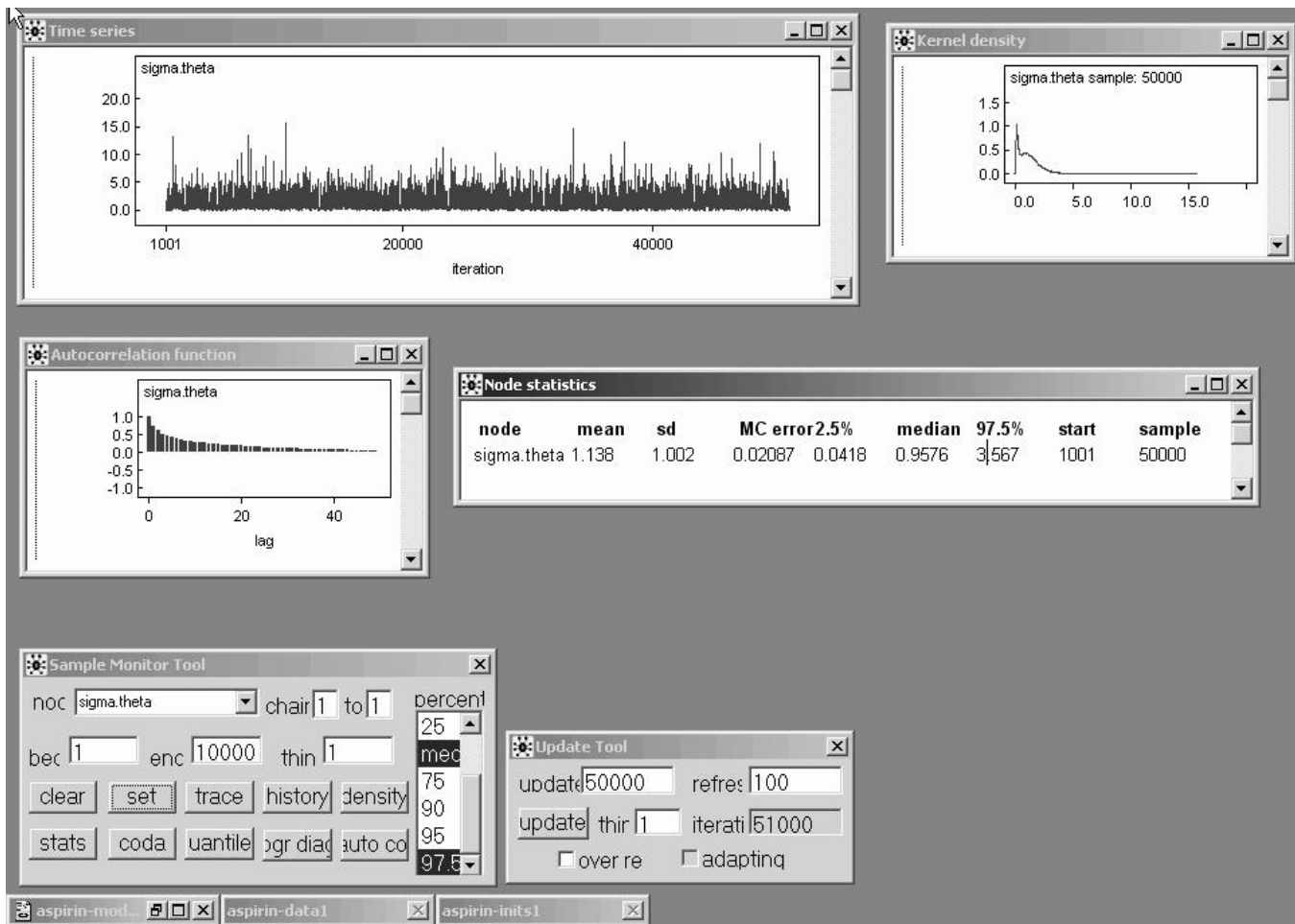
An MCMC sampler which produces output for any given unknown θ with ρ near 0 (if $\rho = 0$ the output is white noise, i.e., equivalent to IID draws from the posterior) is said to be **mixing well** in that unknown.

The time series trace for σ_{θ} above is only mixing **moderately well**: the autocorrelation function has the familiar ski-slope shape of an AR_1 series with $\rho \doteq 0.7$ (the height of the bar at lag 1).

The **marginal posterior distribution** for σ_{θ} (from the Kernel density window) looks heavily skewed to the right, which makes sense for a scale parameter.

The **posterior mean** and **SD** of σ_{θ} (using the $\Gamma(\epsilon, \epsilon)$ prior for τ_{θ}) are estimated to be 1.14 and 1.00, respectively; the **Monte Carlo standard error** of the posterior mean estimate is 0.021 (so that with 50,000 monitoring iterations I don't yet have **3 significant figures** of accuracy for the posterior mean); the **posterior median** is estimated to be 0.96; and a **95% central interval** for σ_{θ} with this prior is estimated to run from 0.042 to 3.57.

WinBUGS Aspirin Analysis (continued)



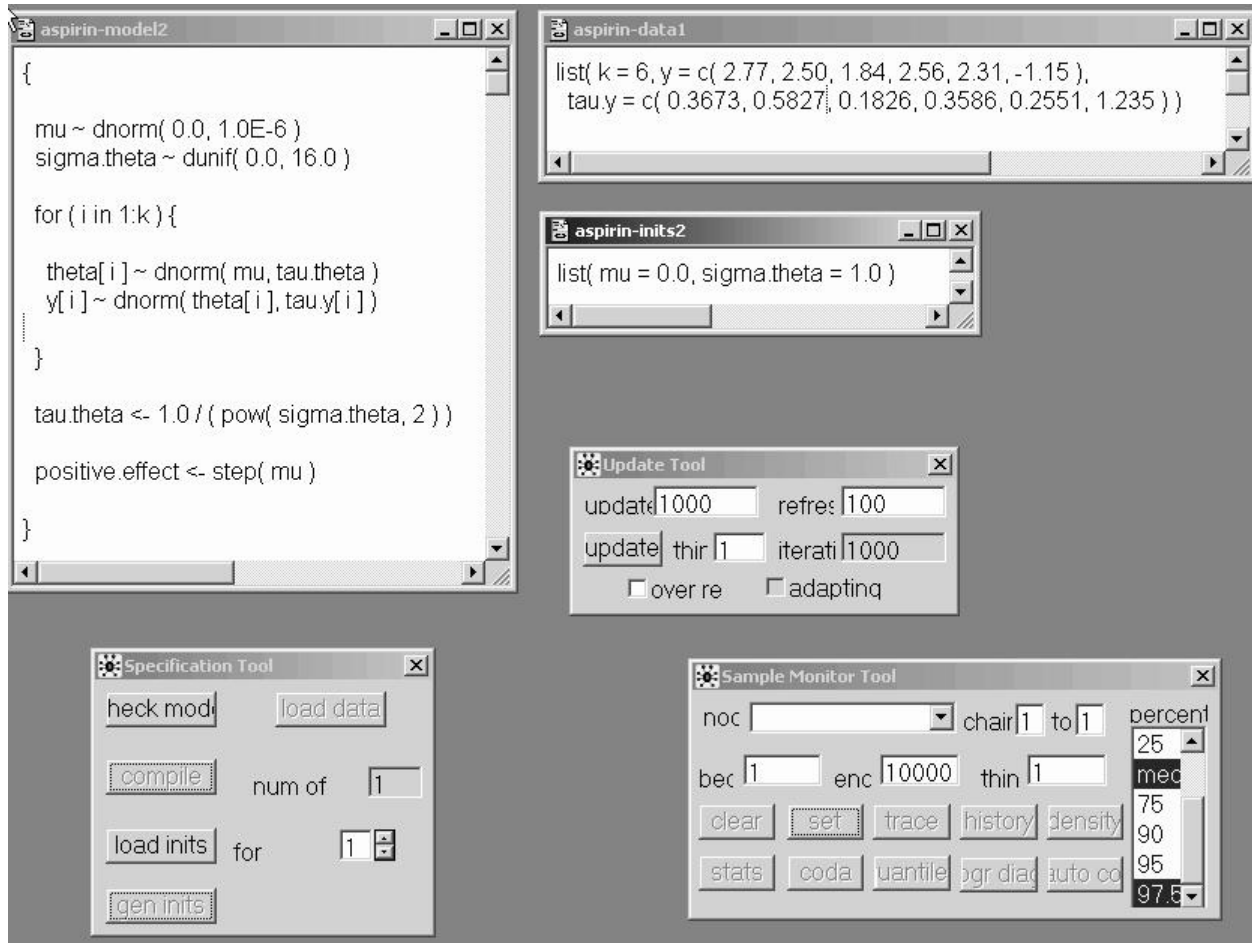
The main thing to notice, however, is that the **range of plausible values** for `sigma.theta` in its posterior is approximately from **0 to 16**.

It has recently been shown that the **simplest diffuse prior** on σ_θ that has **good calibration properties** (i.e., such that **95% nominal** Bayesian interval estimates for all of the parameters in model (10) do in fact have **actual coverage close to 95%**) is

$$\sigma_\theta \sim U(0, c), \quad (10)$$

where c is chosen to be (roughly) the **smallest value that doesn't truncate the likelihood function** for σ_θ ; here it's evident that $c \doteq 16$ will **work well**.

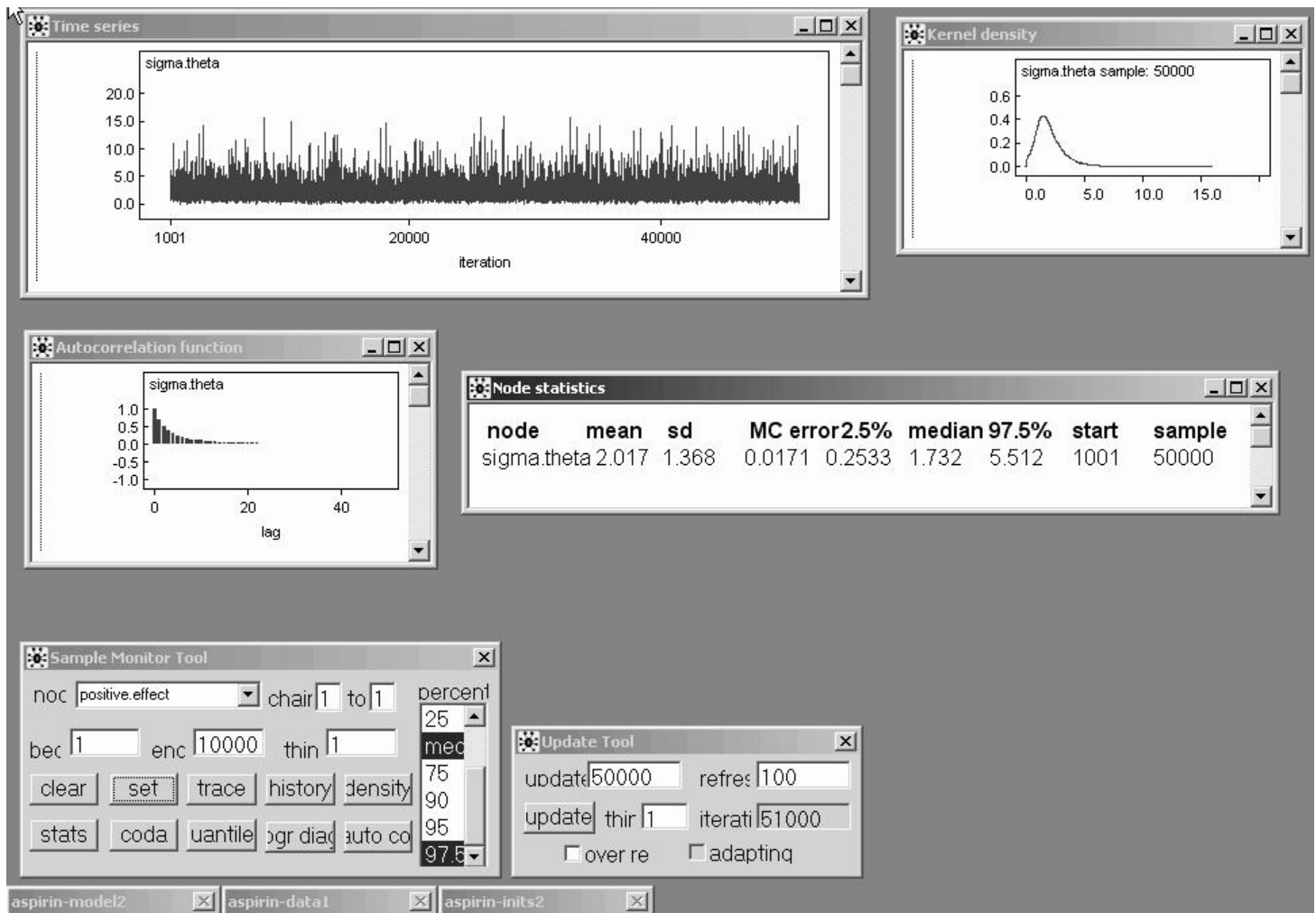
WinBUGS Aspirin Analysis (continued)



So I estimate a **second model** placing a Uniform(0, c) prior on σ_θ (this model also requires a **new initial values file** that initializes `sigma.theta` instead of `tau.theta`).

This time in the Sample Monitor Tool I set all of the **interesting** quantities: `mu`, `sigma.theta`, `theta`, and `positive.effect`, and I use the same MCMC strategy as before (a **burn-in of 1,000** followed by a **monitoring run of 50,000**).

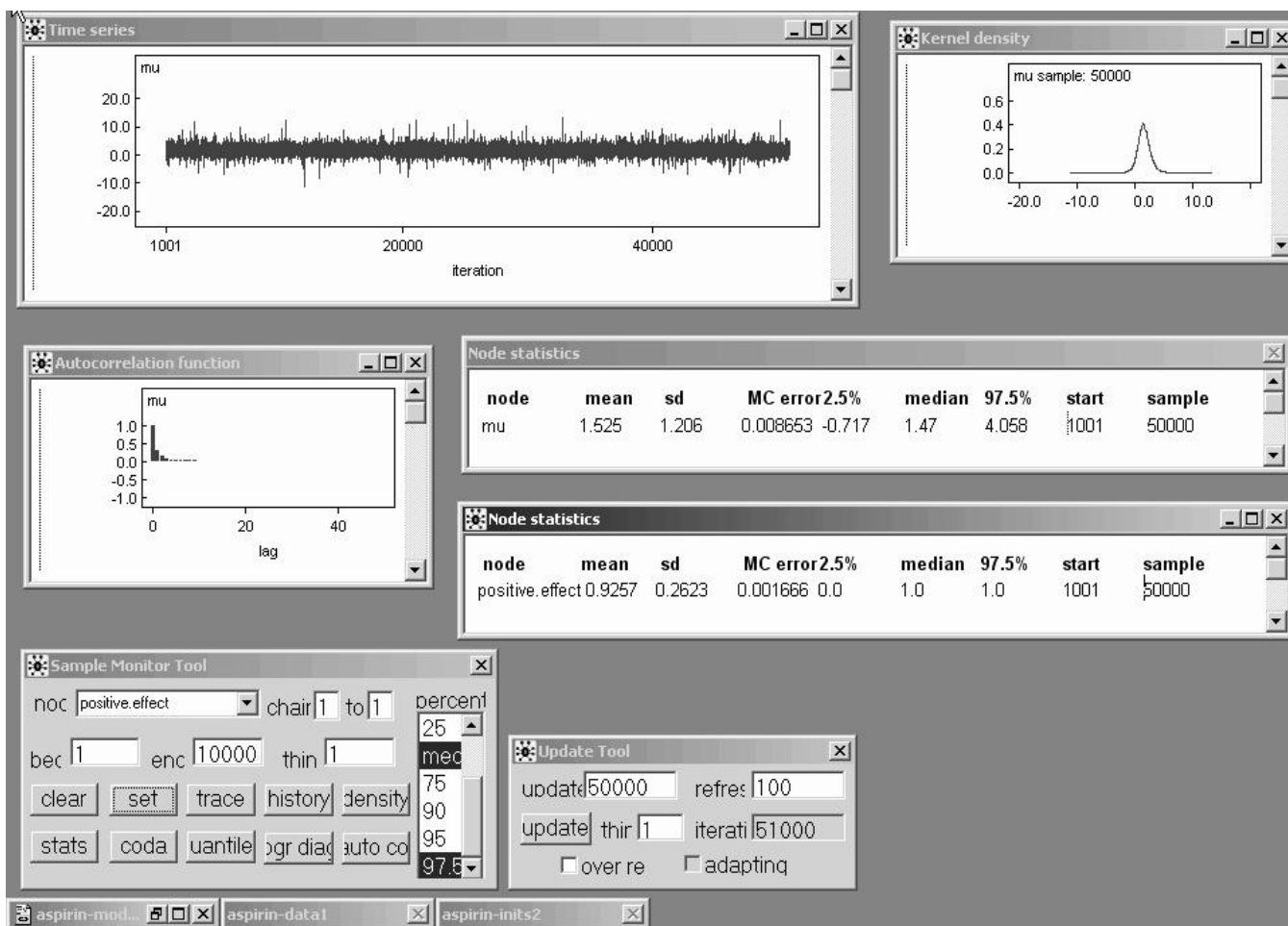
WinBUGS Aspirin Analysis (continued)



With the $\text{Uniform}(0, c)$ prior on σ_θ the posterior mean of σ_θ is now **sharply higher** than before (**2.02** versus the **1.14** value I got with the initial $\Gamma(\epsilon, \epsilon)$ prior (this sort of **discrepancy** will only arise when the number of studies k is **small**; when it does arise I **trust** the results from the $\text{Uniform}(0, c)$ prior).

Note that the posterior mean of σ_θ is also **quite a bit bigger** than the value (**1.24**) obtained from **MLEB** back on page 25—this is a reflection of the **tendency of MLEB to understate the between-study heterogeneity** in model (10) with small k .

WinBUGS Aspirin Analysis (continued)



On pp. 25–26 above we saw that the MLEB estimate of μ was **1.45** with an approximate standard error of **0.809**, and an approximate 95% confidence interval for μ ran from -0.14 to $+3.03$.

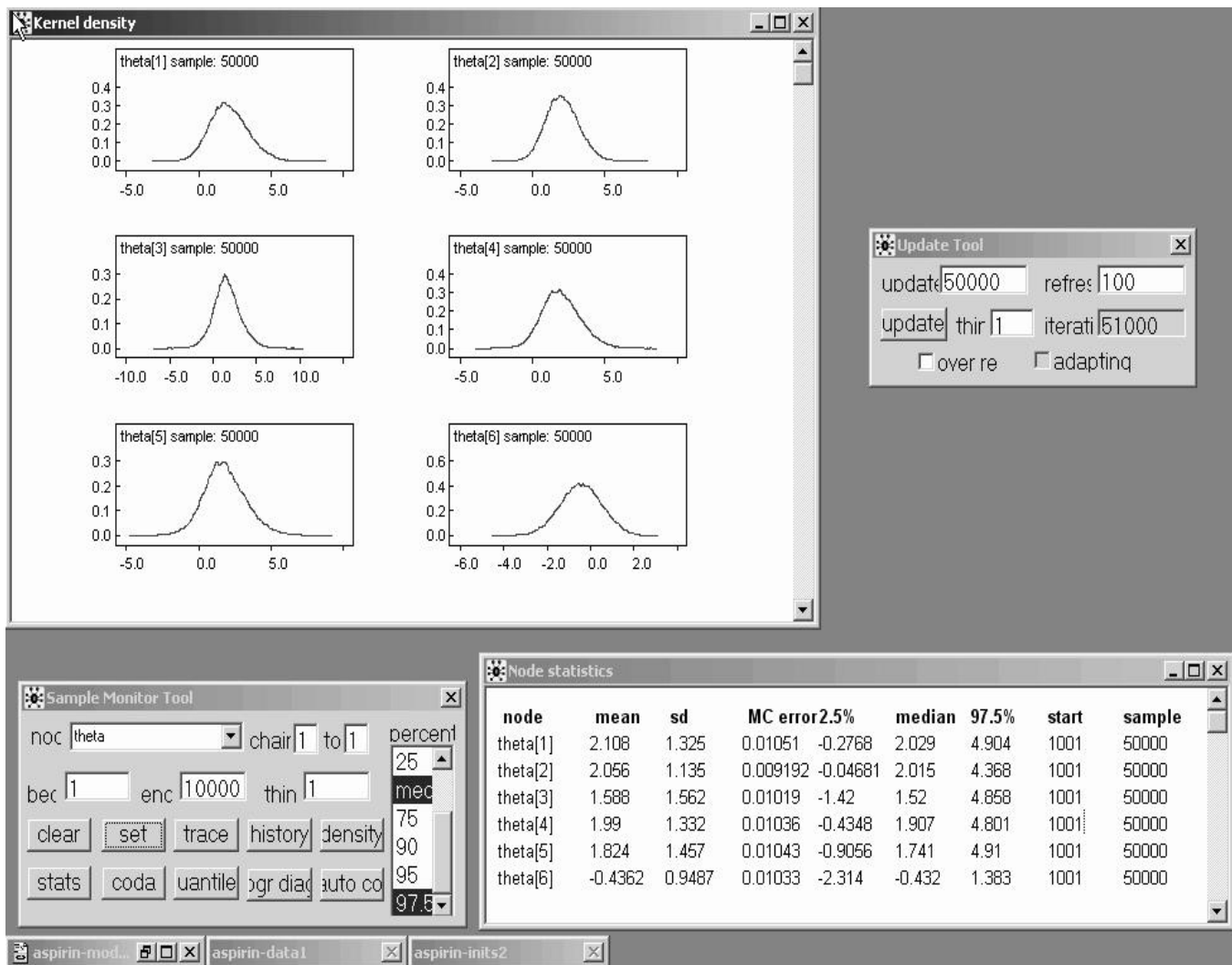
The corresponding **Bayesian** results are: posterior **mean 1.52**, posterior **SD 1.21**, 95% **interval (-0.72, 4.06)**.

As is often true, the simple MLEB approximations leading to these estimates have **underestimated the actual uncertainty** about μ : the Bayesian 95% interval with the Uniform prior is **50% wider**.

It's easy to monitor the **posterior probability that aspirin is beneficial**, with the built-in step function applied to μ :

$P(\mu > 0 | \text{data, diffuse prior information}) \doteq \mathbf{0.93}$, i.e., posterior betting odds of about **12.5 to 1** that **aspirin reduces mortality**.

WinBUGS Aspirin Analysis (continued)



The marginal density plots of the θ_i values show **interesting departures from normality**, and the Bayesian estimates (a) exhibit **rather less shrinkage** and (b) have **27–43% larger uncertainty estimates**.

Table 3.1. MLEB and Bayesian (posterior mean) estimates of the θ_i .

study(i)	Maximum Likelihood		Bayesian Posterior	
	$\hat{\theta}_i$	$\widehat{SE}(\hat{\theta}_i)$	mean	SD
1	1.92	0.990	2.11	1.33
2	1.94	0.899	2.06	1.14
3	1.53	1.09	1.59	1.56
4	1.84	0.994	1.99	1.33
5	1.69	1.05	1.82	1.46
6	-0.252	0.728	-0.44	0.95

Hierarchical Model Expansion

Looking at the **shrinkage plot** on p. 26 or the **raw data values** themselves, it's evident that a **Gaussian** model for the θ_i may not be appropriate: study 6 is so different than the other 5 that a **heavier-tailed distribution** may be a better choice.

This suggests **expanding** the HM (10), by embedding it in a **richer model class** of which it's a **special case** (this is the main Bayesian approach in practice to **dealing with model inadequacies**).

A **natural choice** would be a t model for the θ_i with **unknown degrees of freedom ν** :

$$\begin{aligned}(\theta, \sigma^2, \nu) &\sim p(\theta, \sigma^2, \nu) && \text{(prior)} \\(\theta_i | \theta, \sigma^2, \nu) &\stackrel{\text{IID}}{\sim} t(\theta, \sigma^2, \nu) && \text{(underlying effects)} \\(y_i | \theta_i) &\stackrel{\text{indep}}{\sim} N(\theta_i, V_i) && \text{(data)} .\end{aligned} \quad (11)$$

Here $\eta \sim t(\theta, \sigma^2, \nu)$ just means that $\left(\frac{\eta - \theta}{\sigma}\right)$ follows a **standard t distribution** with ν degrees of freedom. This is **amazingly easy** to implement in WinBUGS (it is considerably more difficult to carry out an **analogous ML analysis**).

The new model file is

```
{

mu ~ dnorm( 0.0, 1.0E-6 )
sigma.theta ~ dunif( 0.0, 16.0 )
nu ~ dunif( 3.0, 30.0 )

for ( i in 1:k ) {

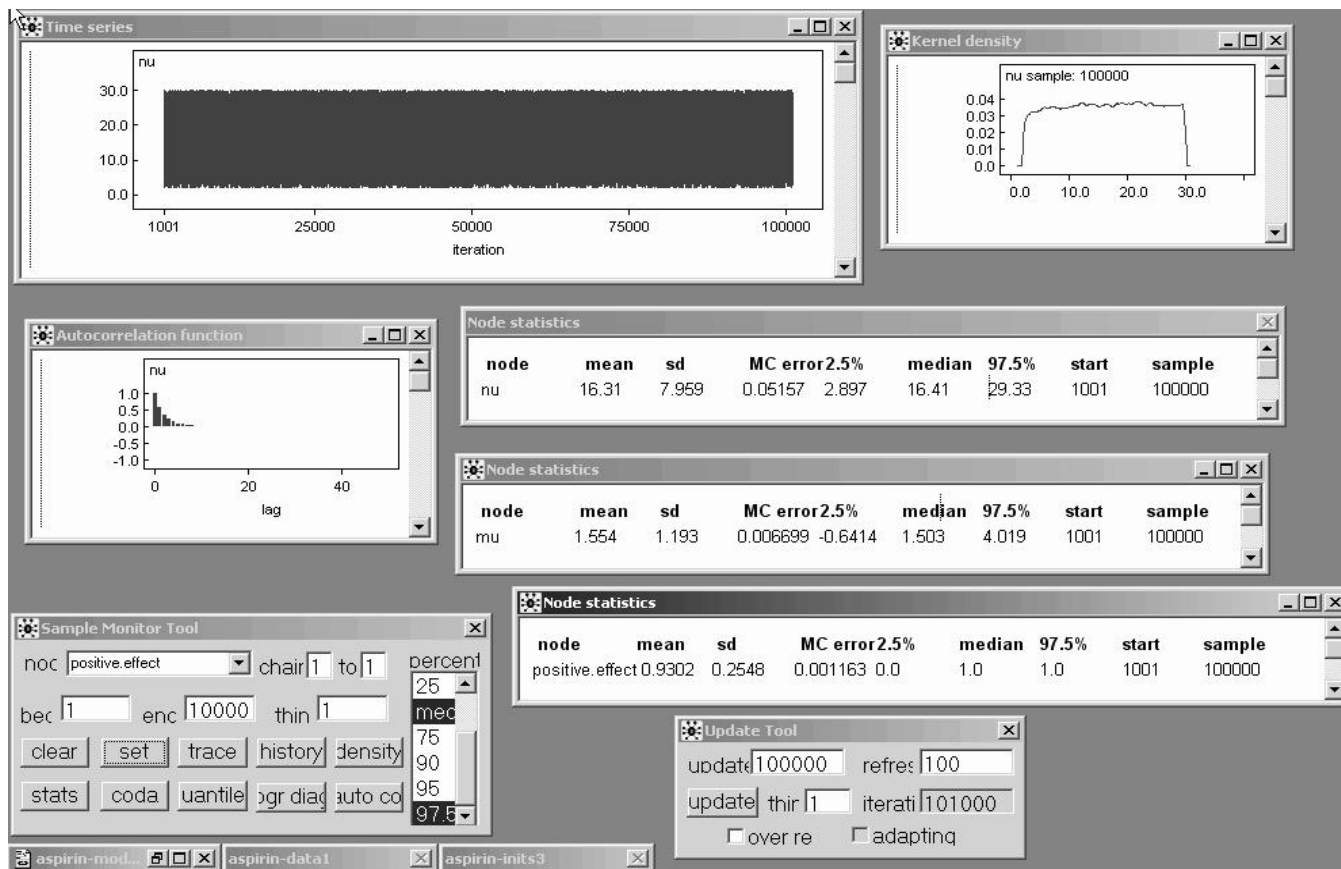
    theta[ i ] ~ dt( mu, tau.theta, nu )
    y[ i ] ~ dnorm( theta[ i ], tau.y[ i ] )

}

tau.theta <- 1.0 / pow( sigma.theta, 2 )

}
```

Model Expansion (continued)



To express comparative prior ignorance about ν I use a **uniform** prior on the interval from 2.0 to 30.0 (below $\nu = 2$ the t distribution has **infinite variance**, and above about 30 it starts to be **indistinguishable** in practice from the Gaussian).

A **burn-in** of 1,000 and a **monitoring run** of 100,000 iterations takes **about twice as long as with 50,000 iterations in the Gaussian model** (i.e., about the **same speed per iteration**) and yields the **posterior summaries** above.

It's clear that there's **little information in the likelihood function** about ν : the prior and posterior for this parameter **virtually coincide**.

The results for μ and the θ_i are **almost unchanged**; this would not necessarily be the case if study 6 had been **more extreme**.

5.3 Hierarchical Model Selection: A Case Study

Case Study: *In-home geriatric assessment (IHGA)*. In an experiment conducted in the 1980s (Hendriksen et al. 1984), 572 elderly people living in a number of villages in Denmark were randomized, 287 to a **control** (*C*) group (who received standard care) and 285 to an **experimental** (*E*) group (who received standard care plus IHGA: a kind of **preventive medicine** in which each person's medical and social needs were assessed and acted upon individually).

One important outcome was the number of **hospitalizations** during the two-year life of the study (Table 6.1).

Table 6.1. Distribution of number of hospitalizations in the IHGA study over a two-year period.

Group	Number of Hospitalizations								<i>n</i>	Mean	SD
	0	1	2	3	4	5	6	7			
Control	138	77	46	12	8	4	0	2	287	0.944	1.24
Experimental	147	83	37	13	3	1	1	0	285	0.768	1.01

Evidently IHGA lowered the mean hospitalization rate (for these elderly Danish people, at least) by $(0.944 - 0.768) = \mathbf{0.176}$, which is about a $100 \left(\frac{0.768 - 0.944}{0.944} \right) = \mathbf{19\%}$ reduction from the control level, a difference that's **large in clinical terms.**

Modeling the IHGA Data

An **off-the-shelf** analysis of this experiment might pretend (**Model 0**) that the data are Gaussian,

$$\begin{aligned} (C_i | \mu_C, \sigma_C^2) &\stackrel{\text{IID}}{\sim} N(\mu_C, \sigma_C^2), i = 1, \dots, n_C, \\ (E_j | \mu_E, \sigma_E^2) &\stackrel{\text{IID}}{\sim} N(\mu_E, \sigma_E^2), j = 1, \dots, n_E, \end{aligned} \quad (12)$$

and use the ordinary frequentist

two-independent-samples “z-machinery”:

```
rosalind 15> R
```

```
R : Copyright 2001, The R Development Core Team  
Version 1.2.1 (2001-01-15)
```

```
> C <- c( rep( 0, 138 ), rep( 1, 77 ), rep( 2, 46 ),  
         rep( 3, 12 ), rep( 4, 8 ), rep( 5, 4 ), rep( 7, 2 ) )
```

```
> print( n.C <- length( C ) )
```

```
[1] 287 # sample size in the control group
```

```
> mean( C )
```

```
[1] 0.9442509 # control group mean
```

```
> sqrt( var( C ) )
```

```
[1] 1.239089 # control group  
# standard deviation (SD)
```

```
> table( C )
```

```
 0  1  2  3  4  5  7 # control group  
138 77 46 12 8 4 2 # frequency distribution
```

Analysis of Model 0

```
> E <- c( rep( 0, 147 ), rep( 1, 83 ), rep( 2, 37 ),
          rep( 3, 13 ), rep( 4, 3 ), rep( 5, 1 ), rep( 6, 1 ) )

> print( n.E <- length( E ) )

[1] 285                # sample size in the
                       # experimental group

> mean( E )

[1] 0.7684211         # experimental group mean

> sqrt( var( E ) )

[1] 1.008268          # experimental group SD

> table( E )

   0  1  2  3  4  5  6    # experimental group
147 83 37 13 3 1 1    # frequency distribution

> print( effect <- mean( E ) - mean( C ) )

[1] -0.1758298        # mean difference ( E - C )

> effect / mean( C )

[1] -0.1862109        # relative difference ( E - C ) / C

> SE.effect <- sqrt( var( C ) / n.C + var( E ) / n.E )

[1] 0.09442807        # standard error of the difference

> print( CI <- c( effect - 1.96 * SE.effect,
                 effect + 1.96 * SE.effect ) )

[1] -0.3609 0.009249  # the 95% confidence interval from
                       # model 0 runs from -.36 to +.01
```


Deficiencies of Model 0

The frequentist analysis of Model 0 is equivalent to a Bayesian analysis of the same model with **diffuse priors** on the control and experimental group means and SDs ($\mu_C, \sigma_C, \mu_E, \sigma_E$), and is summarized in Table 6.2.

Table 6.2. Summary of analysis of Model 0.

	Posterior		
	Mean	SD	95% Interval
Treatment effect ($\mu_E - \mu_C$)	-0.176	0.0944	(-0.361, 0.009)

However, both distributions have long right-hand tails; in fact they look rather **Poisson**.

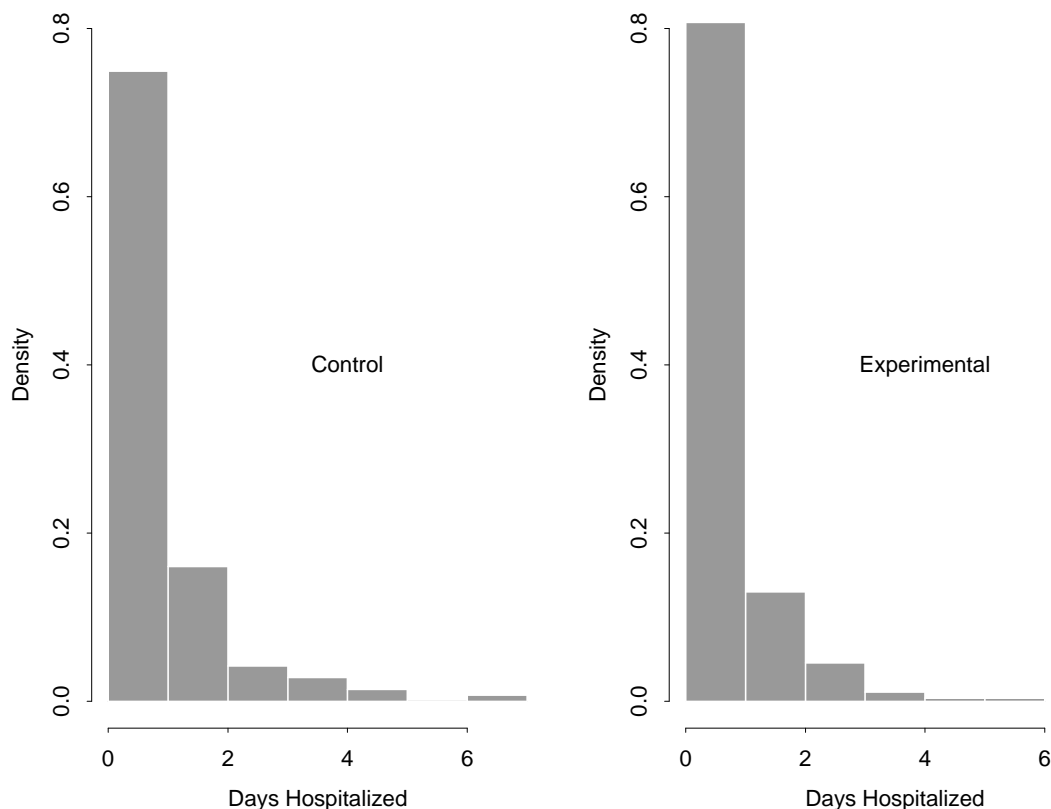


Figure 6.1. Histograms of control and experimental numbers of hospitalizations.

5.4 Poisson Fixed-Effects Modeling

R code to make the histograms:

```
> x11( )                # to open a
                        #   graphics window
> par( mfrow = c( 1, 2 ) )    # to plot two histograms

> hist( C, nclass = 8, probability = T,
      xlab = 'Days Hospitalized', ylab = 'Density',
      xlim = c( 0, 7 ), ylim = c( 0, 0.8 ) )

> text( 4, 0.4, 'Control' )

> hist( E, nclass = 8, probability = T,
      xlab = 'Days Hospitalized', ylab = 'Density',
      xlim = c( 0, 7 ), ylim = c( 0, 0.8 ) )

> text( 4, 0.4, 'Experimental' )
```

So I **created** a classicBUGS file called poisson1.bug that looked like this:

```
model poisson1;

const

  n.C = 287, n.E = 285;

var

  lambda.C, lambda.E, C[ n.C ], E[ n.E ], effect;

data C in "poisson-C.dat", E in "poisson-E.dat";

inits in "poisson1.in";
```

Initial Poisson Modeling (continued)

```
{  
  
lambda.C ~ dgamma( 0.001, 0.001 );  
lambda.E ~ dgamma( 0.001, 0.001 );  
  
for ( i in 1:n.C ) {  
  C[ i ] ~ dpois( lambda.C );  
}  
  
for ( j in 1:n.E ) {  
  E[ j ] ~ dpois( lambda.E );  
}  
  
effect <- lambda.E - lambda.C;  
}
```

poisson1.in initializes both λ_C and λ_E to 1.0; the $\Gamma(0.001, 0.001)$ priors for λ_C and λ_E are chosen (as usual to create diffuseness) to be **flat** in the region in which the **likelihood** is **appreciable**:

```
> sqrt( var( C ) / n.C )
```

```
[1] 0.07314114
```

```
> sqrt( var( E ) / n.E )
```

```
[1] 0.05972466
```

```
> c( mean( C ) - 3.0 * sqrt( var( C ) / n.C ),  
     mean( C ) + 3.0 * sqrt( var( C ) / n.C ) )
```

Initial Poisson Modeling (continued)

```
[1] 0.7248275 1.1636743
```

```
> c( mean( E ) - 3.0 * sqrt( var( E ) / n.E ),  
     mean( E ) + 3.0 * sqrt( var( E ) / n.E ) )
```

```
[1] 0.5892471 0.9475950
```

```
> lambda.grid <- seq( 0.01, 2.0, 0.01 )
```

```
> plot( lambda.grid, 0.001 * dgamma( lambda.grid, 0.001 ),  
       type = 'l', xlab = 'Lambda', ylab = 'Density' )
```

The likelihood under the Gaussian model is **concentrated** for λ_C from about 0.7 to 1.2, and that for λ_E from about 0.6 to 1; you can see from the plot that across those ranges the $\Gamma(0.001, 0.001)$ prior is **essentially constant**.

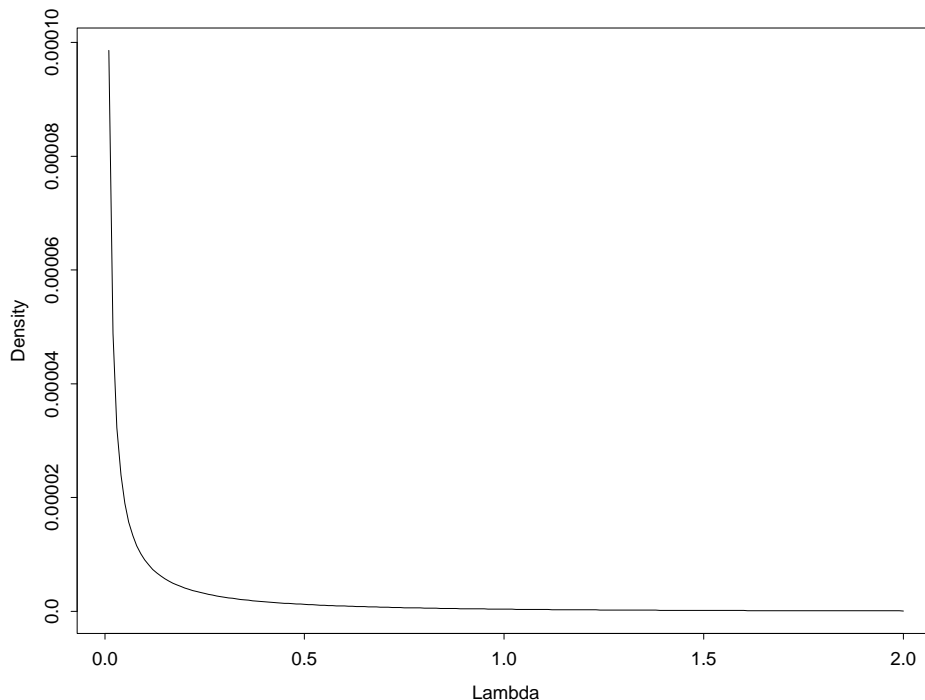
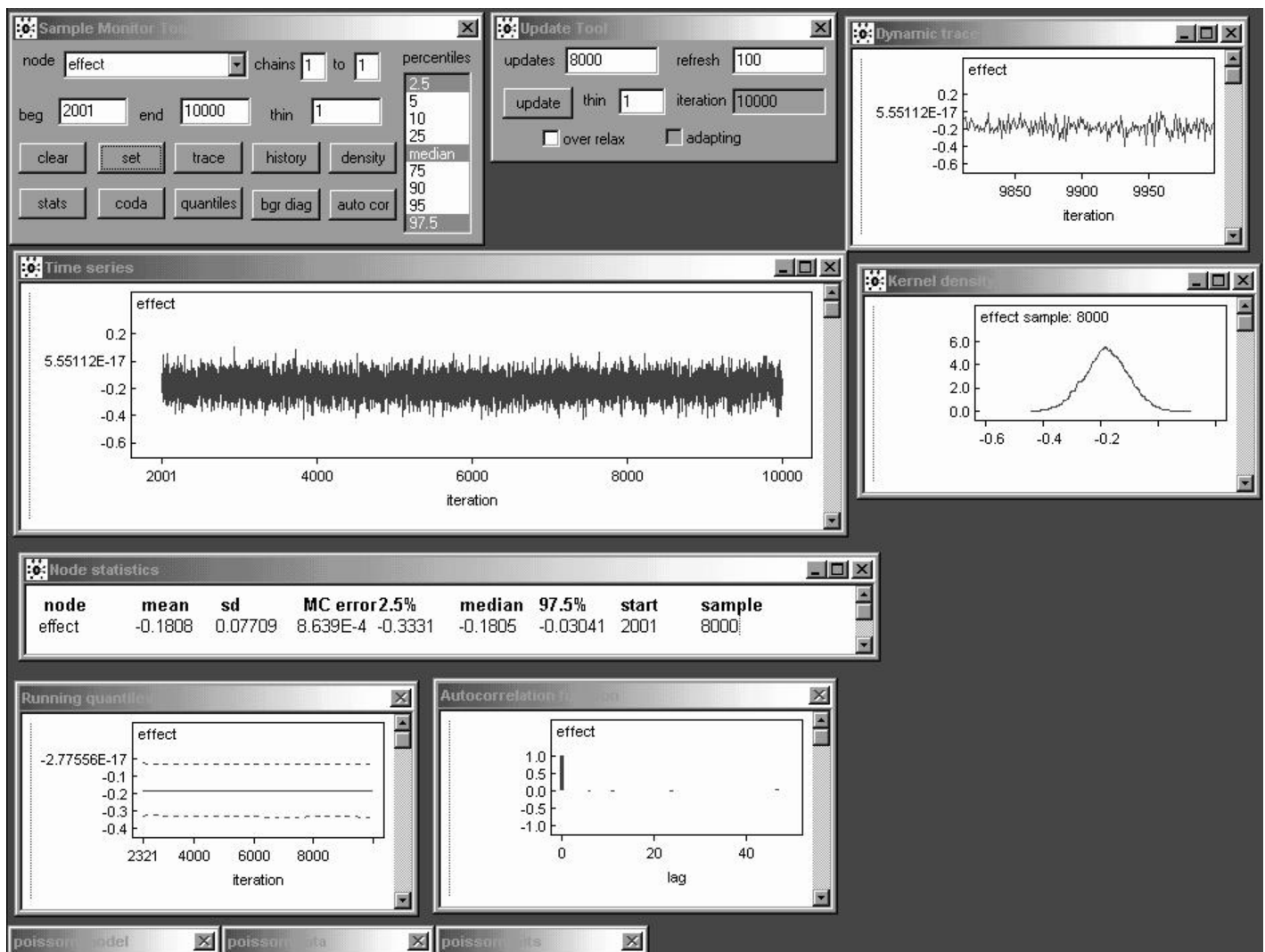


Figure 6.2. The $\Gamma(0.001, 0.001)$ distribution in the region in which the likelihoods for λ_C and λ_E are appreciable.

WinBUGS Implementation (continued)



A monitoring run of 8,000 reveals that the effect parameter in the **2-independent-samples Poisson model** is behaving like **white noise**, so that already with only 8,000 iterations the posterior mean has a Monte Carlo standard error of **less than 0.001**.

Initial Poisson Modeling (continued)

Thus a burn-in of 2,000 and a monitoring run of 8,000 yields **good MCMC diagnostics** and permits a comparison between model 0 (Gaussian) and model 1 (Poisson), as in Table 6.3.

Table 6.3. Comparison of inferential conclusions from models 0 and 1.

λ_C Model	Posterior Mean	Posterior SD	Central 95% Interval
Gaussian	0.944	0.0731	(0.801, 1.09)
Poisson	0.943	0.0577	(0.832, 1.06)

λ_E Model	Posterior Mean	Posterior SD	Central 95% Interval
Gaussian	0.768	0.0597	(0.651, 0.885)
Poisson	0.769	0.0521	(0.671, 0.875)

$\Delta = \lambda_E - \lambda_C$ Model	Posterior Mean	Posterior SD	Central 95% Interval
Gaussian	-0.176	0.0944	(-0.361, 0.009)
Poisson	-0.174	0.0774	(-0.325, -0.024)

The two models produce **almost identical point estimates**, but the Poisson model leads to **sharper inferences** (e.g., the posterior SD for the treatment effect $\Delta = \lambda_E - \lambda_C$ is **22%** larger in model 0 than in model 1).

5.5 Additive and Multiplicative Treatment Effects

This is the same point we noticed with the NB10 data—when a location parameter is the only thing at issue, the Gaussian is a **conservative** modeling choice (intuitively, the Poisson gains its “extra accuracy” from the variance and the mean being equal, which permits **second-moment** information to help in estimating the λ values along with the usual first-moment information).

Both the Gaussian and Poisson models so far implicitly assume that the treatment effect is **additive**:

$$E \stackrel{\text{st}}{=} C + \text{effect}, \quad (13)$$

where $\stackrel{\text{st}}{=}$ means *is stochastically equal to*; in other words, apart from random variation the effect of the IHGA is to **add or subtract a constant** to or from each person’s underlying rate of hospitalization.

However, since the outcome variable is non-negative, it is plausible that a **better model** for the data is

$$E \stackrel{\text{st}}{=} (1 + \text{effect}) C. \quad (14)$$

Additive vs. Multiplicative Effect

Here the treatment effect is **multiplicative**—in other words, apart from random variation the effect of the IHGA is to **multiply** each person's underlying rate of hospitalization by a constant above or below 1.

A **qqplot** of the control and experimental outcome values can in some cases be helpful in choosing between additive and multiplicative models:

```
> CEqq <- qqplot( C, E, plot = F )
```

```
> table( CEqq$y, CEqq$x )
```

```

              Interpolated C values
            0 0.965  1 1.5  2 2.82 3 3.91 4 4.96 5 6.99 7
E 0 137      1  9  0  0      0 0      0 0      0 0      0 0
  1  0      0 66  1 16      0 0      0 0      0 0      0 0
  2  0      0  0  0 29      1 7      0 0      0 0      0 0
  3  0      0  0  0  0      0 4      1 7      1 0      0 0
  4  0      0  0  0  0      0 0      0 0      0 3      0 0
  5  0      0  0  0  0      0 0      0 0      0 0      1 0
  6  0      0  0  0  0      0 0      0 0      0 0      0 1
```

```
> symbols( c( 0, 0.964798, 1, 1, 1.5, 2, 2, 2.823944, 3, 3,
              3.908447, 4, 4.964813, 5, 6.985962, 7 ), c( rep( 0, 3 ),
              rep( 1, 3 ), rep( 2, 3 ), rep( 3, 4 ), 4, 5, 6 ),
           circles = c( 137, 1, 9, 66, 1, 16, 29, 1, 7, 4, 1, 7, 1,
                       3, 1, 1 ), xlab = 'C', ylab = 'E' )
```

Additive vs. Multiplicative Effect

```
> abline( 0, 1 )           # E = C (no effect)
> abline( 0, 0.793, lty = 2 ) # E = 0.816 C
# (multiplicative)
> abline( -0.174, 1, lty = 3 ) # E = C - 0.174 (additive)
```

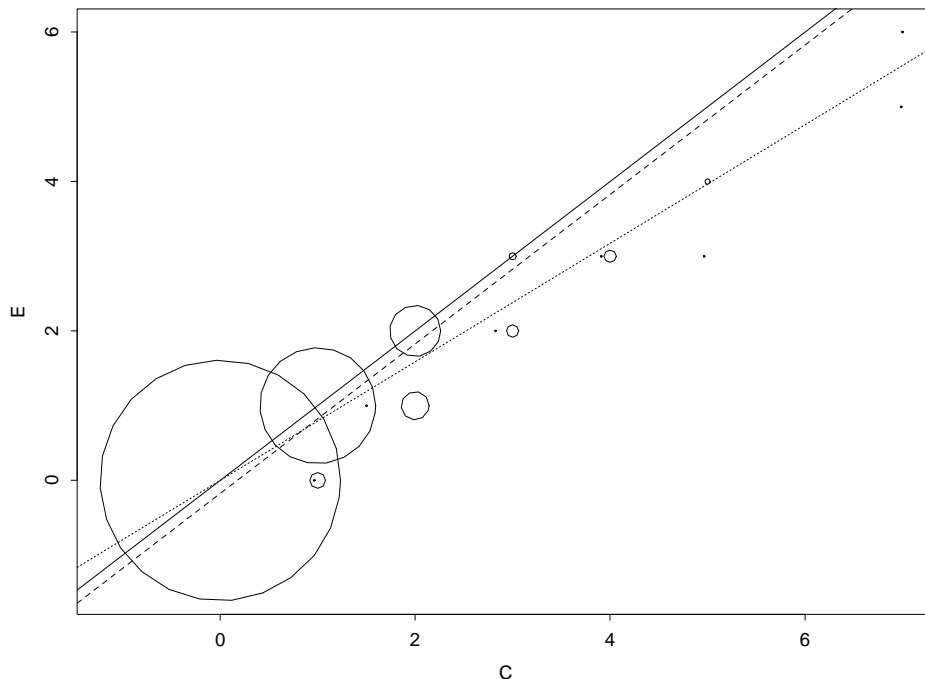


Figure 6.3. QQplot of E versus C values, with the radii of the plotted circles proportional to the number of observations at the indicated point. The solid line corresponds to no treatment effect, the small dotted line to the best-fitting multiplicative model ($E \stackrel{\text{st}}{=} 0.816 C$), and the large dotted line to the best-fitting additive model ($E \stackrel{\text{st}}{=} C - 0.174$).

Here, because the Poisson model has only **one parameter** for both location and scale, the multiplicative and additive formulations **fit equally well**, but the multiplicative model **generalizes more readily** (see below).

A Multiplicative Poisson Model

A simple way to write the multiplicative model is to re-express the data in the form of a **regression** of the outcome y on a **dummy variable** x which is 1 if the person was in the experimental group and 0 if he/she was in the control group:

i	1	2	...	287	288	289	...	572
x_i	0	0	...	0	1	1	...	1
y_i	1	0	...	2	0	3	...	1

Then for $i = 1, \dots, n = 572$ the **multiplicative** model can be written

$$\begin{aligned}
 (y_i | \lambda_i) &\stackrel{\text{indep}}{\sim} \text{Poisson}(\lambda_i) \\
 \log(\lambda_i) &= \gamma_0 + \gamma_1 x_i \\
 (\gamma_0, \gamma_1) &\sim \text{diffuse}
 \end{aligned} \tag{15}$$

In this model the **control** people have

$$\log(\lambda_i) = \gamma_0 + \gamma_1(0) = \gamma_0, \quad \text{i.e.,} \quad \lambda_C = e^{\gamma_0}, \tag{16}$$

and the **experimental** people have

$$\begin{aligned}
 \log(\lambda_i) &= \gamma_0 + \gamma_1(1) = \gamma_0 + \gamma_1, \quad \text{i.e.,} \\
 \lambda_E &= e^{\gamma_0 + \gamma_1} = e^{\gamma_0} e^{\gamma_1} = \lambda_C e^{\gamma_1}.
 \end{aligned} \tag{17}$$

Now you may remember from basic **Taylor series** that for γ_1 not too far from 0

$$e^{\gamma_1} \doteq 1 + \gamma_1, \tag{18}$$

A Multiplicative Poisson Model

so that finally (for γ_1 fairly near 0)

$$\lambda_E \doteq (1 + \gamma_1) \lambda_C, \quad (19)$$

which is a way of expressing equation (3) in **Poisson language**.

Fitting this model in classicBUGS is easy:

```
model poisson2;

const

  n = 572;

var

  gamma.0, gamma.1, lambda[ n ], x[ n ], y[ n ], lambda.C,
  lambda.E, mult.effect;

data x in "poisson-x.dat", y in "poisson-y.dat";
inits in "poisson2.in";

{
  gamma.0 ~ dnorm( 0.0, 1.0E-4 );      # flat priors for
  gamma.1 ~ dnorm( 0.0, 1.0E-4 );      # gamma.0 and gamma.1

  for ( i in 1:n ) {

    log( lambda[ i ] ) <- gamma.0 + gamma.1 * x[ i ];
    y[ i ] ~ dpois( lambda[ i ] );

  }

  lambda.C <- exp( gamma.0 );
  lambda.E <- exp( gamma.0 + gamma.1 );
  mult.effect <- exp( gamma.1 );
}
```

WinBUGS Implementation (continued)

The screenshot displays the WinBUGS interface with several windows open:

- poisson2data**: A window showing the data input list. The first list, `list(y = c(...))`, contains 572 values ranging from 0 to 6. The second list, `x = c(...)`, contains 572 values ranging from 0 to 6. The total number of observations is `n = 572`.
- Update Tool**: A control panel with fields for `updates` (2000) and `refresh` (100). It includes `update` and `thin` (1) buttons, and checkboxes for `over relax` and `adapting`.
- poisson2mcmc**: A window showing the MCMC list: `list(gamma.0 = 0.0, gamma.1 = 0.0)`.
- poisson2model**: A window showing the model specification code:

```
{
  gamma.0 ~ dnorm( 0.0, 1.0E-4 )
  gamma.1 ~ dnorm( 0.0, 1.0E-4 )

  for( i in 1:n ){
    log( lambda[ i ] ) <- gamma.0 +
      gamma.1 * x[ i ]
    y[ i ] ~ dpois( lambda[ i ] )
  }

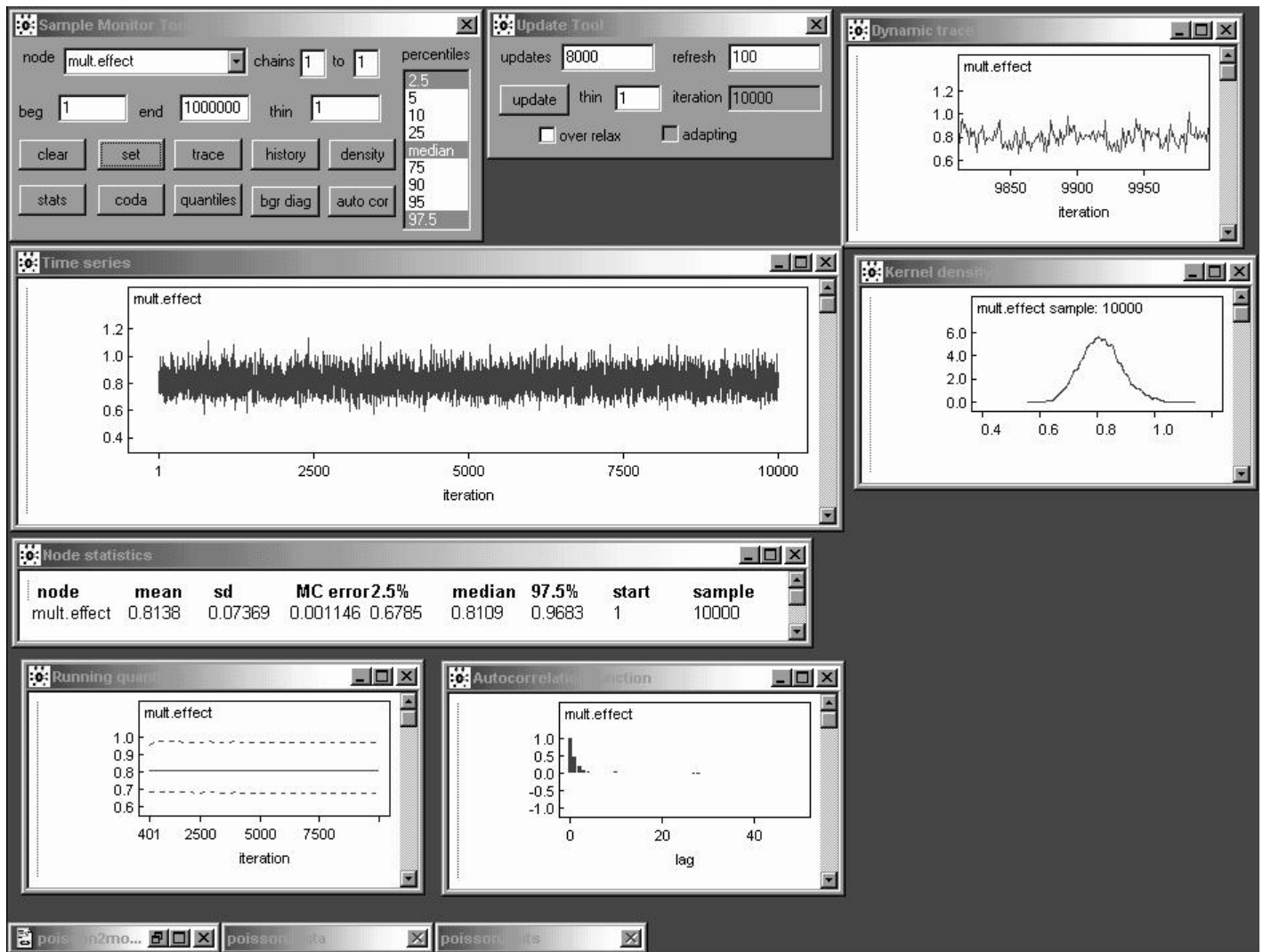
  lambda.C <- exp( gamma.0 )
  lambda.E <- exp( gamma.0 + gamma.1 )
  mult.effect <- exp( gamma.1 )
}
```
- Sample Monitor Tool**: A control panel for monitoring the MCMC process. It shows the selected node `mult.effect` and a table of percentiles:

percentiles
2.5
5
10
25
median
75
90
95
97.5
- Specification Tool**: A control panel with buttons for `check model`, `load data`, `compile`, `load inits`, and `gen inits`. It also includes a `num of chains` field set to 1 and a `for chain` field set to 1.

At the bottom left, a status bar indicates `updates took 26 s`.

The **multiplicative Poisson model** (4) takes longer to run—2,000 burn-in iterations now take about **4 seconds at 2.0 PC GHz**—but still exhibits **fairly good mixing**, as we’ll see below.

WinBUGS Implementation (continued)



A total of **10,000 iterations** (the chain started essentially in equilibrium, so the burn-in can be absorbed into the monitoring run) reveals that the **multiplicative effect parameter** e^{γ_1} in model (4) behaves like an AR_1 series with $\hat{\rho}_1 \doteq 0.5$, but the Monte Carlo standard error for the posterior mean is still only about **0.001** with a run of this length.

Additive versus Multiplicative Fit

A burn-in of 2,000 and a monitoring run of 8,000 again yields **good MCMC diagnostics** and permits a comparison between the additive and multiplicative Poisson models, as in Table 6.4.

Table 6.4. Comparison of inferential conclusions from the additive and multiplicative Poisson models.

λ_C Model	Posterior Mean	Posterior SD	Central 95% Interval
additive	0.943	0.0577	(0.832, 1.06)
multiplicative	0.945	0.0574	(0.837, 1.06)

λ_E Model	Posterior Mean	Posterior SD	Central 95% Interval
additive	0.769	0.0521	(0.671, 0.875)
multiplicative	0.768	0.0518	(0.671, 0.872)

effect Model	Posterior Mean	Posterior SD	Central 95% Interval
additive	-0.174	0.0774	(-0.325, -0.024)
multiplicative	-0.184	0.0743	(-0.324, -0.033)

With this model it is as if the experimental people's average underlying rates of hospitalization have been **multiplied by 0.82**, give or take about 0.07.

The additive and multiplicative effects are **similar** here, because both are not too far from zero.

Extra-Poisson Variability

However, none of this has verified that the **Poisson model is reasonable** for these data—the histograms show that the Gaussian model is clearly unreasonable, but the diagnostic plots in WinBUGS and CODA only check on the adequacy of the **MCMC** sampling, not the model.

In fact we had a good clue that the data are **not** Poisson back on page 2: as noted in part 2, the Poisson(λ) distribution has mean λ and also variance λ —in other words, the **variance-to-mean-ratio** (VTMR) for the Poisson is 1. But

```
> var( C ) / mean( C )  
[1] 1.62599  
> var( E ) / mean( E )  
[1] 1.322979
```

i.e., the data exhibit **extra-Poisson variability** (VTMR > 1).

This actually **makes good sense** if you think about it, as follows.

The Poisson model assumes that everybody in the control group has the **same underlying rate** λ_C of hospitalization, and similarly everybody in the experimental group has the **same rate** λ_E .

Unobserved Predictor Variables

In reality it's far more reasonable to think that each person has his/her **own** underlying rate of hospitalization that depends on **baseline health status, age**, and various other things.

Now Hendriksen **forgot to measure** (or at least to report on) these other variables (he may have hoped that the randomization would balance them between C and E)—the only predictor we have is x , the **experimental status dummy variable**—so the best we can do is to lump all of these other **unobserved** predictor variables together into a kind of **“error” term** e .

This amounts to **expanding** the second Poisson model (4) above: for $i = 1, \dots, n = 572$ the new model is

$$\begin{aligned} (y_i | \lambda_i) &\stackrel{\text{indep}}{\sim} \text{Poisson}(\lambda_i) \\ \log(\lambda_i) &= \gamma_0 + \gamma_1 x_i + e_i \\ e_i &\stackrel{\text{IID}}{\sim} N(0, \sigma_e^2) \\ (\gamma_0, \gamma_1, \sigma_e^2) &\sim \text{diffuse.} \end{aligned} \quad (20)$$

5.6 Random- Effects Poisson Regression

The **Gaussian** choice for the error distribution is **conventional**, not dictated by the science of the problem (although if there were a lot of unobserved predictors hidden inside the e_i their weighted sum would be close to normal by the **Central Limit Theorem**).

Model (9) is an **expansion** of the earlier model (4) because you can obtain model (4) from (9) by setting $\sigma_e^2 = 0$, whereas with (9) we're letting σ_e^2 vary and **learning about it from the data**.

The addition of the **random effects** e_i to the model is one way to address the extra-Poisson variability: this model would be called a **lognormal mixture of Poisson distributions** (or a **random effects Poisson regression** (REPR) model) because it's as if each person's λ is drawn from a lognormal distribution and then his/her number of hospitalizations y is drawn from a Poisson distribution with his/her λ , and this mixing process will make the variance of y **bigger than its mean**.

WinBUGS Implementation

The new WinBUGS model is

```
{  
  
  gamma.0 ~ dnorm( 0.0, 1.0E-4 )  
  gamma.1 ~ dnorm( 0.0, 1.0E-4 )  
  tau.e ~ dgamma( 0.001, 0.001 )  
  
  for ( i in 1:n ) {  
  
    e[ i ] ~ dnorm( 0.0, tau.e )  
    log( lambda[ i ] ) <- gamma.0 + gamma.1 * x[ i ] +  
      e[ i ]  
    y[ i ] ~ dpois( lambda[ i ] )  
  
  }  
  
  lambda.C <- exp( gamma.0 )  
  lambda.E <- exp( gamma.0 + gamma.1 )  
  mult.effect <- exp( gamma.1 )  
  sigma.e <- 1.0 / sqrt( tau.e )  
  
}
```

I again use a **diffuse** $\Gamma(\epsilon, \epsilon)$ prior (with $\epsilon = 0.001$)
for the **precision** τ_e of the random effects.

Sensitivity to Initial Values

Warning WinBUGS can fail, particularly in random-effects models, when you give it initial values that are not very close to the final posterior means; an example in release 1.3 is the REPR model (9) on the IHGA data with the “**generic**” starting values $(\gamma_0, \gamma_1, \tau_e) = (0.0, 0.0, 1.0)$.

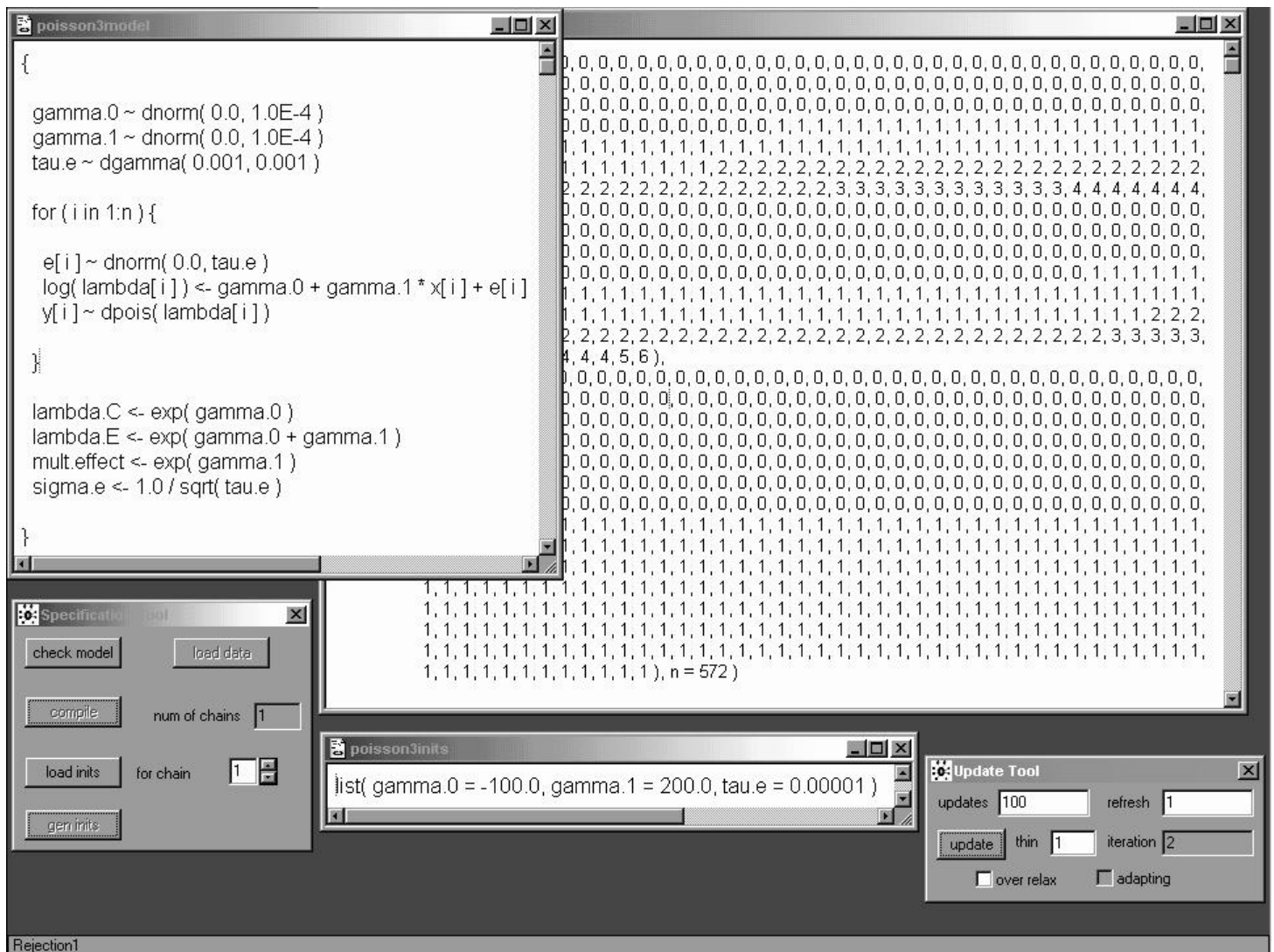
When this problem arises there are two ways out in WinBUGS: **trial and error**, or a calculation (see below).

NB MLwiN does not have this problem—it gets its starting values from **maximum likelihood** (the mode of the likelihood function is often a decent approximation to the mean or mode of the posterior).

Technical note. To get a decent starting value for τ_e in model (9) you can calculate as follows: renaming the random effects η_i to avoid confusion with the number e ,

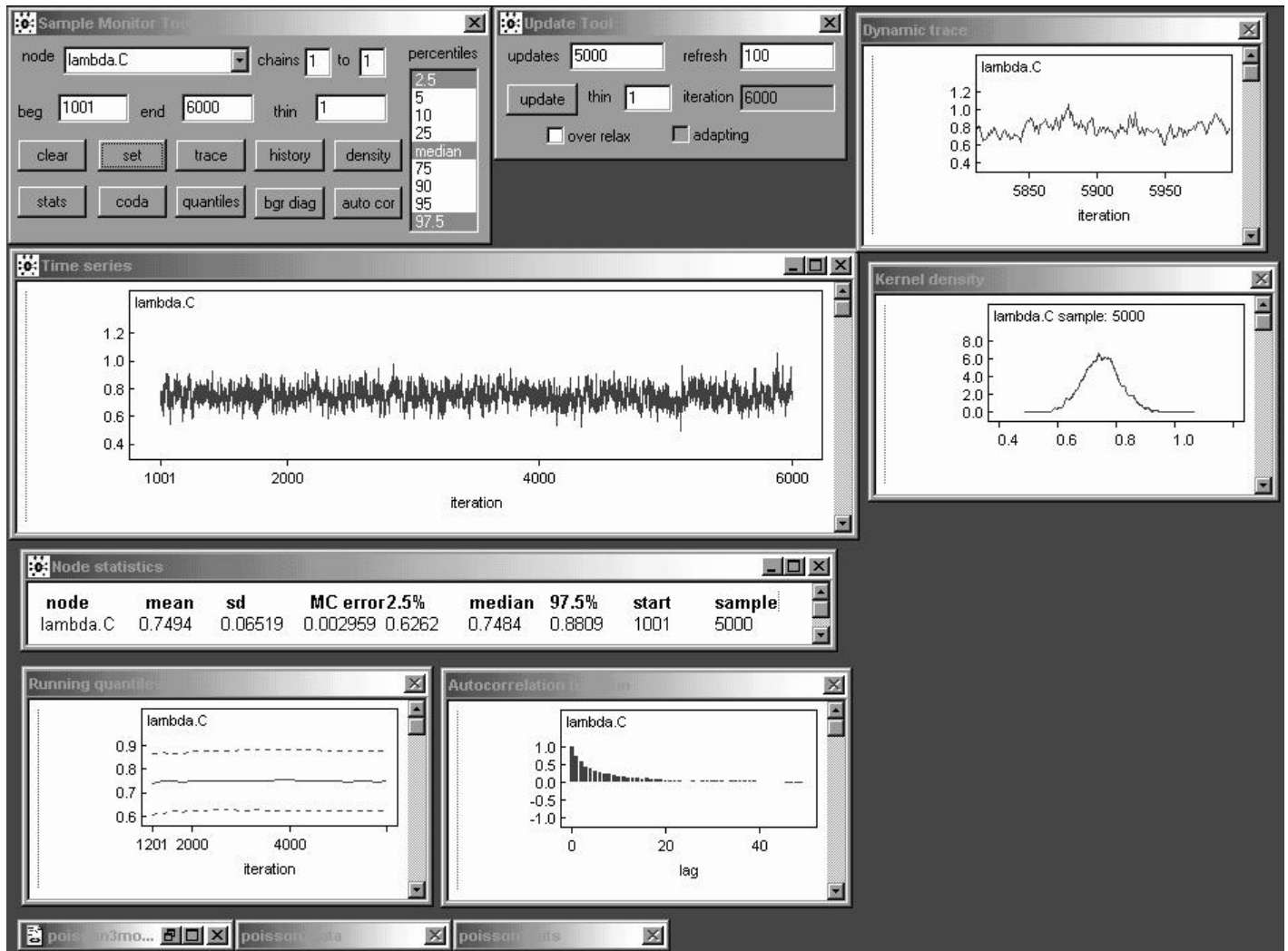
(1) $V(y_i) = V[E(y_i | \eta_i)] + E[V(y_i | \eta_i)]$, where
(2) $(y_i | \eta_i) \sim \text{Poisson}(e^{\gamma_0 + \gamma_1 x_i + \eta_i})$, so
 $E(y_i | \eta_i) = V(y_i | \eta_i) = e^{\gamma_0 + \gamma_1 x_i + \eta_i}$. Then (3)
 $V[E(y_i | \eta_i)] = V(e^{\gamma_0 + \gamma_1 x_i + \eta_i}) = e^{2(\gamma_0 + \gamma_1 x_i)} V(e^{\eta_i})$ and
 $E[V(y_i | \eta_i)] = E(e^{\gamma_0 + \gamma_1 x_i + \eta_i}) = e^{\gamma_0 + \gamma_1 x_i} E(e^{\eta_i})$. Now (4) e^{η_i} is lognormal with mean 0 and variance σ_e^2 on the log scale, so
 $E(e^{\eta_i}) = e^{\frac{1}{2}\sigma_e^2}$ and $V(e^{\eta_i}) = e^{\sigma_e^2} (e^{\sigma_e^2} - 1)$, yielding finally
 $V(y_i) = e^{2(\gamma_0 + \gamma_1 x_i) + \frac{1}{2}\sigma_e^2} + e^{\gamma_0 + \gamma_1 x_i + \sigma_e^2} (e^{\sigma_e^2} - 1)$. (5) Plugging in $x_i = 0$ for the C group, whose sample variance is 1.54, and using the value $\gamma_0 = -0.29$ from runs with previous models, gives an equation for σ_e^2 that can be solved numerically, yielding $\sigma_e^2 \doteq 0.5$ and $\tau_e \doteq 2$.

WinBUGS Implementation (continued)



Interestingly, WinBUGS release 1.4 is able to **sample successfully** with the generic starting values $(\gamma_0, \gamma_1, \tau_e) = (0.0, 0.0, 1.0)$, although of course a **longer burn-in period** would be needed when they're used; you have to try **truly absurd** initial values to get it to fall over, and when it does so the **error message** ("Rejection1") in the lower left corner is **more discreet**.

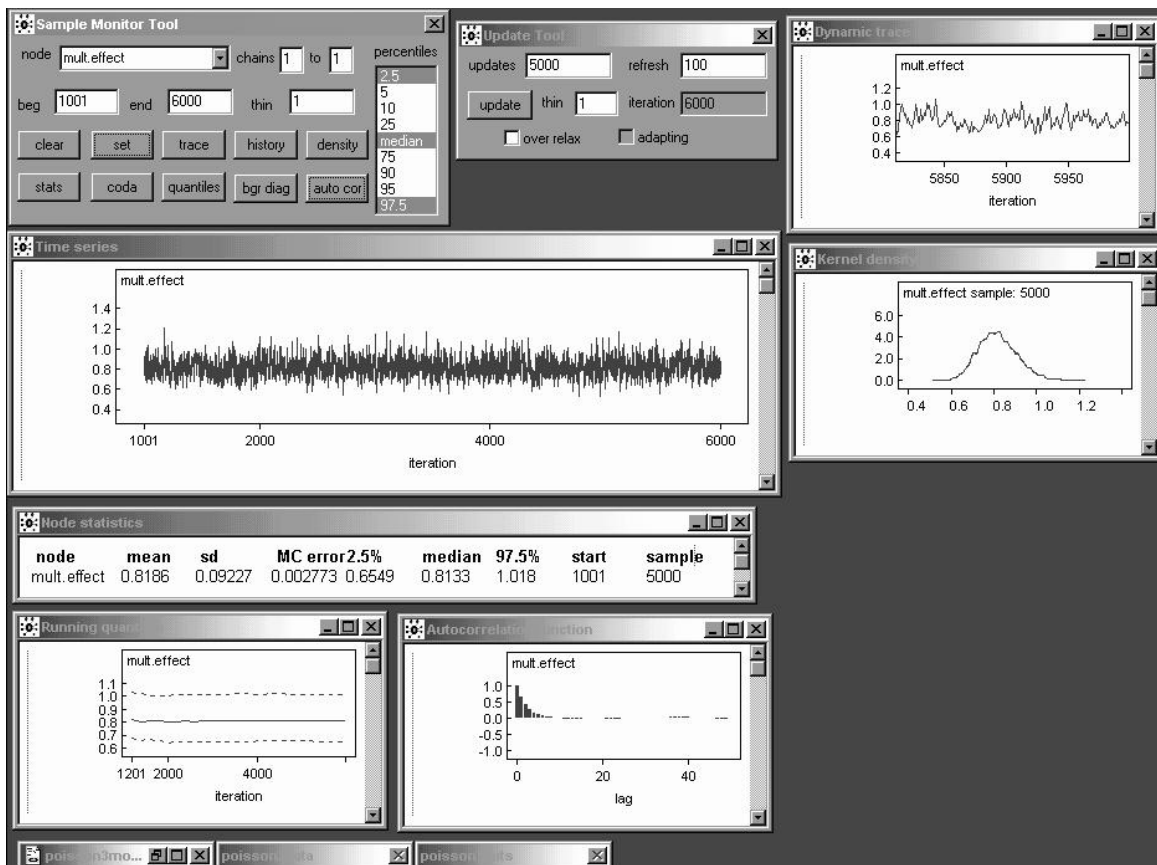
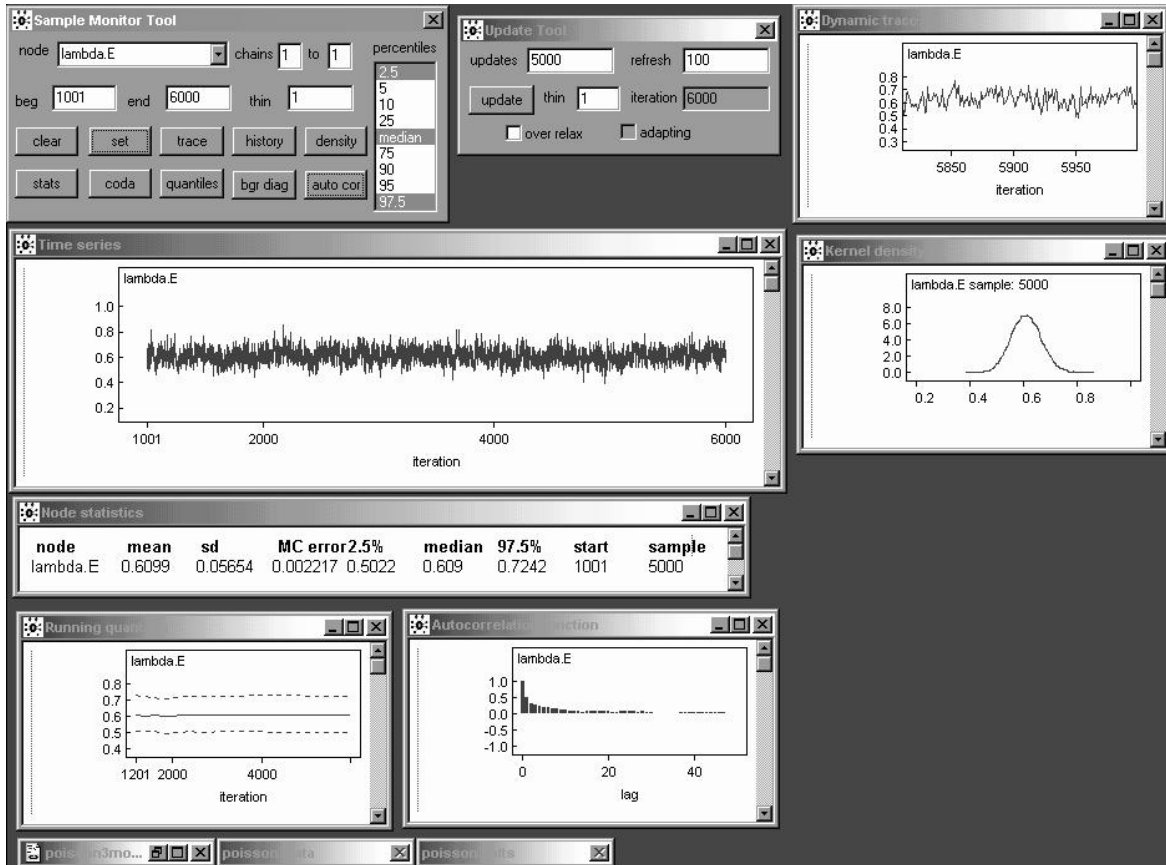
WinBUGS Implementation (continued)



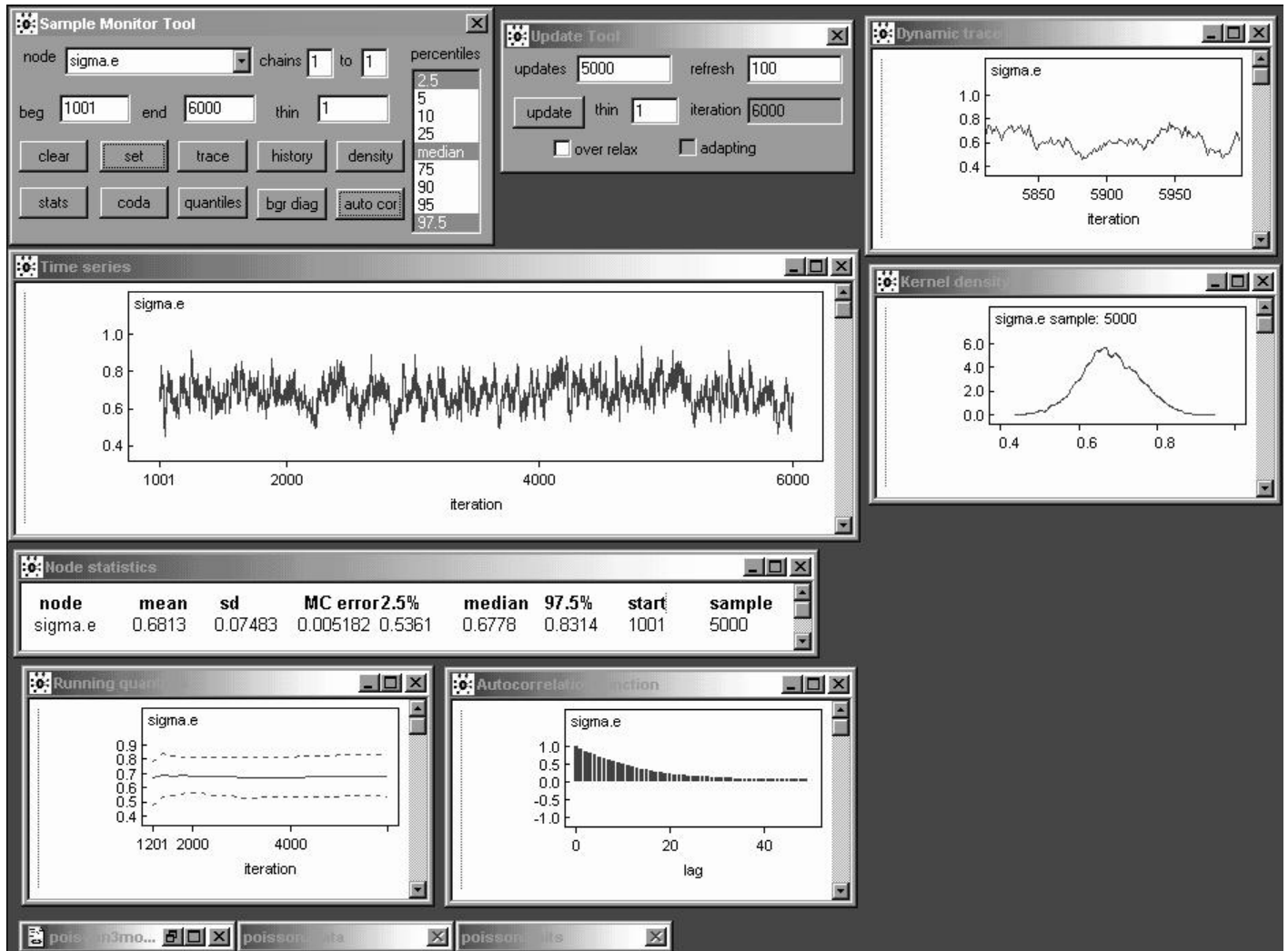
With a **better set of initial values**— $(\gamma_0, \gamma_1, \tau_e) = (-0.058, -0.21, 2.0)$, obtained from (a) the earlier Poisson models (in the case of the regression parameters γ_j) and (b) either a calculation like the one on the bottom of page 25 or trial and error—WinBUGS is able to make progress, although this model takes a **fairly long time to fit** in release 1.4: a burn-in of 1,000 takes 5.5 seconds at 2.0 PC GHz (for some reason the code runs about twice as fast in release 1.3).

A monitoring run of **5,000** iterations reveals that the random effects make everything **mix more slowly**: λ_C (this page) and λ_E and the multiplicative effect (next page) all behave like AR_1 series with $\hat{\rho}_1 \doteq 0.7, 0.5, \text{ and } 0.6$, respectively.

WinBUGS Implementation (continued)



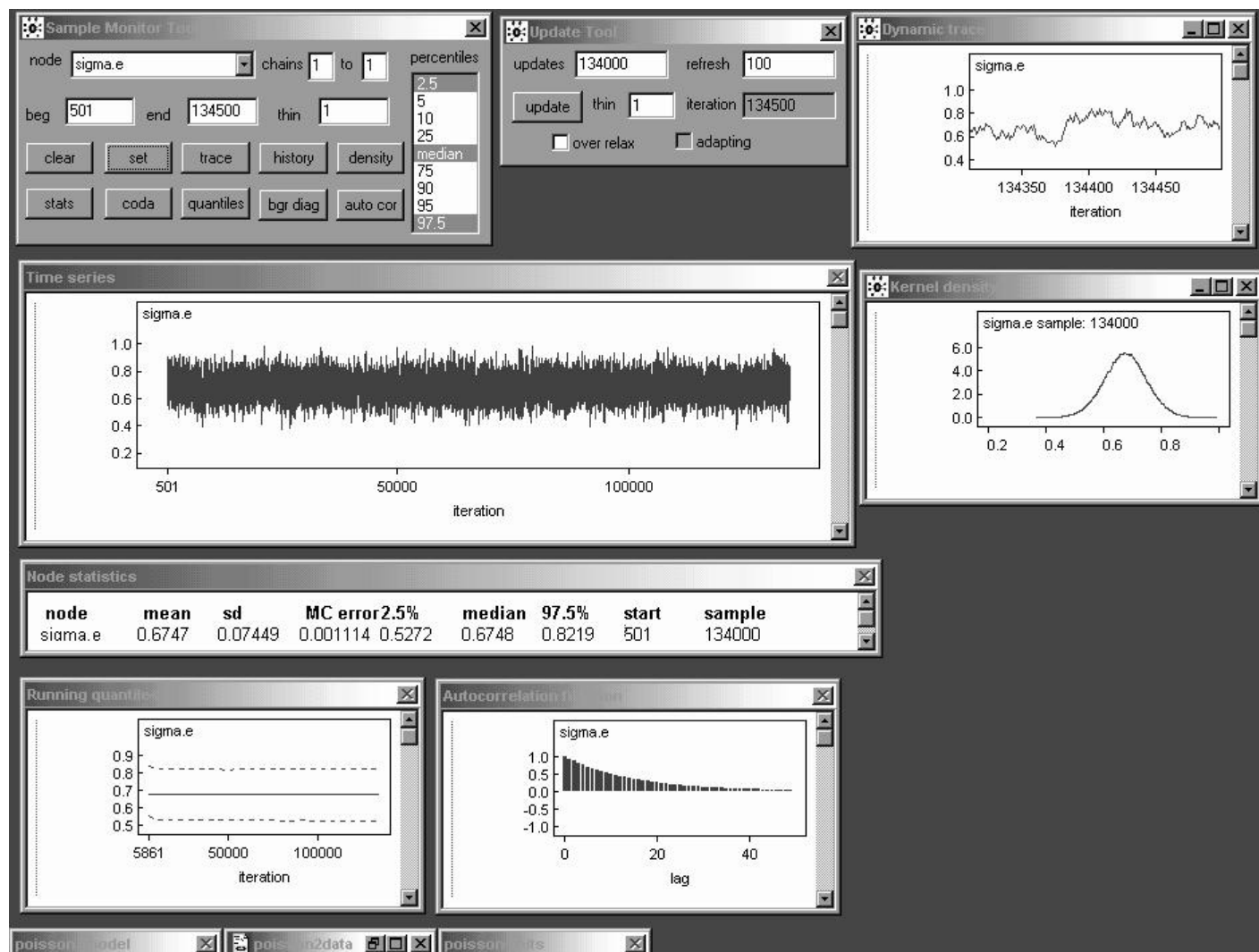
WinBUGS Implementation (continued)



Learning about σ_e in this model is **slow**: its autocorrelation function is that of an AR_1 with a **high value** of $\hat{\rho}_1$ (equation (55) on page 75 of part 3 gives $\hat{\rho}_1 \doteq \mathbf{0.92}$).

The MCSE of the posterior mean for σ_e based on 5,000 draws is **0.005182**—to get this down to (say) **0.001** I need to increase the length of the monitoring run by a factor of $\left(\frac{0.005182}{0.001}\right)^2 \doteq \mathbf{26.9}$, meaning a total run of about $(26.9)(5,000) \doteq \mathbf{134,000}$ iterations (this takes about **15 minutes** at 2.0 PC GHz).

WinBUGS Implementation (continued)



There is **clear evidence** that σ_e is **far from 0**—its posterior mean and SD are estimated as **0.675** (with an MCSE of about **0.001** after 134,000 iterations) and **0.074**, respectively—meaning that the **model expansion** from (4) to (9) was **amply justified**.

REPR Model Results

(Another way to achieve the goal of describing the extra-Poisson variability would be to fit different **negative binomial** distributions to the observed counts in the C and E groups—the negative binomial is a **gamma mixture of Poissons**, and the gamma and lognormal distributions often fit long-tailed data about equally well, so you would not be surprised to find that the two approaches give **similar results**.)

Table 6.5. Comparison of inferential conclusions about the multiplicative effect parameter e^{γ_1} from the fixed- and random-effects Poisson regression models.

Model	Posterior Mean	Posterior SD	Central 95% Interval
FEPR	0.816	0.0735	(0.683, 0.969)
REPR	0.830	0.0921	(0.665, 1.02)

Table 6.5 compares the REPR model inferential results with those from model (4), which could also be called a **fixed-effects Poisson regression** (FEPR) model.

The “error” SD σ_e has posterior mean **0.68**, give or take about 0.07 (on the $\log(\lambda)$ scale), corresponding to substantial extra-Poisson variability, which translates into **increased uncertainty** about the multiplicative effect parameter e^{γ_1} .

I’ll argue later that the REPR model **fits the data well**, so the conclusion I’d publish from these data is that IHGA reduces the average number of hospitalizations per two years by about $100(1 - 0.083)\% = \boxed{17\%}$ give or take about 9% (ironically this conclusion is similar to that from the Gaussian model, but this is **coincidence**).

Two More Items on MCMC Accuracy

(1) A **stringent** but potentially **useful** diagnostic for deciding how long the **monitoring run** should be for a given component θ' of the parameter vector θ , if the output of your MCMC sampler for θ' behaves like an AR_1 series with first-order **autocorrelation** ρ_1 , can be derived as follows.

Suppose, after a **burn-in** that's long enough to reach **stationarity**, you've done a **preliminary** monitoring run, obtaining mean $\bar{\theta}'$, SD $\hat{\sigma}_{\theta'}$, and first-order **autocorrelation** $\hat{\rho}_1$ as **estimates** of the corresponding summaries for θ' .

Writing $\theta' = a \cdot 10^b$ for $1 \leq a < 10$, if you want at least k **significant figures** (sigfigs) of accuracy for the posterior mean summary for θ' with **Monte Carlo probability** of at least $100(1 - \alpha)$, you can check that you'll need

$$2\Phi^{-1}\left(1 - \frac{\alpha}{2}\right) \widehat{SE}(\bar{\theta}') \leq 10^{b-k+1}; \quad (21)$$

then **substituting** in the relevant expression from equation (51) in part 3,

$$\widehat{SE}(\bar{\theta}') = \frac{\hat{\sigma}_{\theta'}}{\sqrt{m}} \sqrt{\frac{1 + \hat{\rho}_1}{1 - \hat{\rho}_1}}, \quad (22)$$

and **solving** (12) for m yields

$$m \geq 4 \left[\Phi^{-1}\left(1 - \frac{\alpha}{2}\right) \right]^2 \left(\frac{\hat{\sigma}_{\theta'}}{10^{b-k+1}} \right)^2 \left(\frac{1 + \hat{\rho}_1}{1 - \hat{\rho}_1} \right). \quad (23)$$

This is referred to in the MLwiN documentation as the **Brooks-Draper** diagnostic (Brooks and Draper 2004).

Comments. (a) This diagnostic is **sensitive** to the **scale** chosen by the user for reporting results, as far as choosing the **target** number of sigfigs is concerned.

MCMC Accuracy (continued)

Example. In my initial monitoring run of 5,000 iterations in the **NB10** case study, the **posterior mean** of μ , on the micrograms below 10g scale, was $\bar{\theta}' = 404.3$ (to 4 sigfigs); the other relevant quantities for μ were as follows: **posterior SD** $\hat{\sigma}_{\theta'} \doteq 0.464$ and **first-order autocorrelation** $\hat{\rho}_1 \doteq 0.294$ (**NB** the MCSE for μ is already down to **0.009** with 5,000 iterations, so I already have a bit more than **4 sigfigs** of accuracy).

Suppose (just for the sake of **illustration**; it's hard to imagine setting an accuracy goal this stringent in practice) that I want to ensure **5 sigfigs** with at least 95% Monte Carlo probability for the posterior mean—write $\bar{\theta}' = 4.043 \cdot 10^2$, so that $b = 2$, take $\alpha = 0.05$ and **substitute** into (14) to yield

$$m \geq 4(1.96)^2 \left(\frac{0.464}{10^{2-5+1}} \right)^2 \left(\frac{1 + 0.294}{1 - 0.294} \right) \doteq 60,600. \quad (24)$$

Now, if you instead **subtracted 404** from all of the data values (on the micrograms below 10g scale) and made a similar MCMC run, everything would be the same as above except that your current posterior mean for μ would be **0.3** to 1 sigfig, and (with the same MCSE of 0.009) you would regard yourself as already having a bit more than **1 sigfig** of accuracy from the initial monitoring run of 5,000.

Then to apply (14) to get **2 sigfigs** of accuracy you would write $\bar{\theta}' = 3.0 \cdot 10^{-1}$ and obtain

$$m \geq 4(1.96)^2 \left(\frac{0.464}{10^{(-1)-2+1}} \right)^2 \left(\frac{1 + 0.294}{1 - 0.294} \right) \doteq 60,600. \quad (25)$$

These two sets of results from (14) are **consistent**—by subtracting 404 from all of the data values you (at least temporarily) **threw away 3 sigfigs**—but you can see that **care** needs to be taken in thinking about how much **accuracy** you want, and this question is closely tied to the **scale of measurement**.

(b) Note from (14) that every time you want to add **1 new sigfig** of accuracy in the posterior mean the required length of monitoring run **goes up multiplicatively** by $(10^1)^2 = 100$.

MCMC Accuracy (continued)

(2) I've concentrated so far on the **MCMC accuracy** of the **posterior mean**—what about other posterior summaries like the **SD**?

Suppose as above that you're interested in a given component θ' of the parameter vector θ , and that the output of your MCMC sampler for θ' behaves like an AR_1 series with first-order **autocorrelation** ρ_1 ; and suppose as above that after a **burn-in** that's long enough to reach **stationarity**, you've done a **preliminary** monitoring run, obtaining mean $\bar{\theta}'$, SD $\hat{\sigma}_{\theta'}$, and first-order **autocorrelation** $\hat{\rho}_1$ as **estimates** of the corresponding summaries for θ' .

Then it can be shown, in an expression **analogous** to (13), that if the **marginal posterior** for θ' is approximately **Gaussian**

$$\widehat{SE}(\hat{\sigma}_{\theta'}) = \frac{\hat{\sigma}_{\theta'}}{\sqrt{2m}} \sqrt{\frac{1 + \hat{\rho}_1^2}{1 - \hat{\rho}_1^2}}. \quad (26)$$

Note that with a **parameter** with MCMC output that's approximately AR_1 and **roughly Gaussian** this implies that

$$\frac{\widehat{SE}(\bar{\theta}')}{\widehat{SE}(\hat{\sigma}_{\theta'})} \doteq \sqrt{\frac{2(1 + \hat{\rho}_1)^2}{1 + \hat{\rho}_1^2}}, \quad (27)$$

which goes from $\sqrt{2}$ to 2 as $\hat{\rho}_1$ ranges from 0 to +1, i.e., the **mean is harder to pin down than the SD with Gaussian data** (a reflection of how **light** the tails are).

5.7 References

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