The Use of Rejection Odds and Rejection Ratios in Testing Hypotheses

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Contemporary Issues in Hypothesis Testing

CRiSM

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The sad state of statistical testing in science

- Significance testing using p-values is by far the dominant method of testing in science.
- Its standard uncritical use is viewed by many as being the major source of the problems of reproducibility of science.
- Everyone is talking about it:
  - articles in all the major science journals;
  - changes in editorial policy (the journal *Basic and Applied Social Psychology* banned p-values);
  - the recent ASA position statement about p-values and discussion.
- My view: none of the discussion matters unless, as a profession, we can agree on an alternative to p-values.

The p-value Controversy

- Concerns with use of p-values trace back to at least Berkson (1937).
- Concerns are recurring in many scientific literatures:
  Environmental sciences: http://www.indiana.edu/~stigtsts/
  Social sciences: http://acs.tamu.edu/~bbt6147/
  Wildlife science: http://www.npwrc.usgs.gov/perm/hypotest/
  http://www.cnr.colostate.edu/~anderson/null.html
- An example - articles and books in social sciences: Rozeboom, 60; Edwards, Lindman, and Savage, 63; Morrison and Henkel, 70; Carver, 78; Meehl, 78; Shaver, 85; Oakes, 86; Kupersmid, 88; Rosnow and Rosenthal, 89; Cohen, 90, 94; Rosenthal, 91; Thompson, 93, 94, 98, 99; Volume 51 (No.4) of J. of Experimental Education; Schmidt, 96 (APA presidential address); Hunter, 97; Schmidt and Hunter, 97; Harlow, Mulaik and Steiger, 97; Levin, 98; APA task force
- Numerous works specifically focus on comparing the Fisher and N-P approaches (e.g., Lehmann, 1993 JASA: The Fisher, N-P Theories of Testing Hypotheses: One Theory or Two?)

The Major Problem: p-values are misinterpreted

- Few non-statisticians understand p-values, most erroneously thinking they are some type of error probability, Bayesian or frequentist; they are neither!
  - A survey 30 years ago:
    - "What would you conclude if a properly conducted, randomized clinical trial of a treatment was reported to have resulted in a beneficial response ($p < 0.05$)?
      1. Having obtained the observed response, the chances are less than 5% that the therapy is not effective.
      2. The chances are less than 5% of not having obtained the observed response if the therapy is effective.
      3. The chances are less than 5% of having obtained the observed response if the therapy is not effective.
      4. None of the above.
    - We asked this question of 24 physicians ... Half ... answered incorrectly, and all had difficulty distinguishing the subtle differences...
    - The correct answer to our test question, then, is 3."

"This isn't right. This isn't even wrong." –Wolfgang Pauli, on a submitted paper

* Actual correct answer: The chances are less than 5% of having obtained the observed response ***or any more extreme response*** if the therapy is not effective.

- But, is it fair to count 'possible data more extreme than the actual data' in the evidence against the null hypothesis?

  Jeffreys (1961): "An hypothesis, that may be true, may be rejected because it has not predicted observable results that have not occurred."

- Matthews (1998): "The plain fact is that 70 years ago Ronald Fisher gave scientists a mathematical machine for turning baloney into breakthroughs, and flukes into funding."

- When testing precise hypotheses, true error probabilities (Bayesian or frequentist) are much larger than p-values.
  - Later examples.
  - *Applet* (of Jarad Niemi) at https://jaradniemi.shinyapps.io/pvalue/

Replacing p-values and fixed error probability frequentist testing with unified Bayesian/frequentist rejection odds

- Review of why Bayesian hypothesis testing and the common usage of p-values are incompatible.
- Review of why Bayesian hypothesis testing and fixed error probability frequentist testing are incompatible.
- Why use of rejection odds can unify Bayesian and frequentist testing.

A Key Issue: Is the precise hypothesis being tested plausible?

A *precise hypothesis* is an hypothesis of lower dimension than the alternative, $H_0 : \mu = 0$ versus $H_1 : \mu \neq 0$ (or $H_0 : |\mu| < \epsilon$ versus $H_1 : |\mu| > \epsilon$).

A precise hypothesis is *plausible* if it has a reasonable prior probability of being true. H_0 : "the Higgs boson has spin 0" *is* plausible.

Example: Let θ denote the difference in mean treatment effects for cancer treatments A and B, and test $H_0 : \theta = 0$ versus $H_1 : \theta \neq 0$.

Scenario 1: Treatment A = standard chemotherapy
Treatment B = standard chemotherapy + steroids

Scenario 2: Treatment A = standard chemotherapy
Treatment B = a new radiation therapy

$H_0 : \theta = 0$ is plausible in Scenario 1, but not in Scenario 2; in the latter case, instead test $H_0 : \theta < 0$ versus $H_1 : \theta > 0$.

Plausible precise null hypotheses:

- H_0 : Gene A is not associated with Disease B.
- H_0: There is no psychokinetic effect.
- H_0: Vitamin C has no effect on the common cold.
- H_0: A new HIV vaccine has no effect.
- H_0: Cosmic microwave background radiation is isotropic.
- H_0 : Males and females have the same distribution of eye color.
- H_0 : Pollutant A does not cause disease B.

Implausible precise null hypotheses:

- H_0 : Small mammals are as abundant on livestock grazing land as on non-grazing land
- H_0 : Bird abundance does not depend on the type of forest habitat they occupy
- H_0 : Children of different ages react the same to a given stimulus.

Bayesian hypothesis testing and the common usage of p-values are incompatible

San Jose Mercury News

mercurynews.com WEST VALLEY 102 Friday, September 25, 2009 THE NEWSPAPER OF SILICON VALLEY 75 cents

AIDS MILESTONE

New path for HIV vaccine

Some in study protected from infection, but trial raises more questions

By Karen Kaplan and Thomas H. Maugh II
Los Angeles Times

Hours after HIV researchers announced the achievement of a milestone that had eluded them for a quarter of a century, reality began to set in: Tangible progress could take another decade.

A Thai and American team announced early Thursday in Bangkok that they had found a combination of vaccines providing modest protection against infection with the virus that causes AIDS, unleashing excitement worldwide. The idea of a vaccine to prevent infection with the human immunodeficiency virus, HIV, had long been frustrating and fruitless.

But by Thursday afternoon, initial euphoria gave way to a more sober assessment. There is still a very long way to go before reaching the goal of producing a vaccine that reliably shields people from HIV.

Some researchers questioned whether the apparent 31 percent reduction in infections was a sta-

See **VACCINE**, *Page 14*

A researcher during the Thai phase III HIV Vaccine Trial, also known as RV 144, tests the "prime-boost" combination of two vaccines.

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Hypotheses and data:

- Alvac had shown no effect
- Aidsvax had shown no effect

Question: Would Alvac as a primer and Aidsvax as a booster work?

The Study: Conducted in Thailand with 16,395 individuals from the general (not high-risk) population:

- 74 HIV cases reported in the 8198 individuals receiving placebos
- 51 HIV cases reported in the 8197 individuals receiving the treatment

The test that was performed:

- Let p_1 and p_2 denote the probability of HIV infection in the placebo and treatment populations, respectively.
- Test $H_0 : p_1 = p_2$ versus $H_1 : p_1 > p_2$
- Normal approximation okay, so

$$z = \frac{\hat{p}_1 - \hat{p}_2}{\sqrt{\hat{\sigma}_{\{\hat{p}_1 - \hat{p}_2\}}}} = \frac{.009027 - .006222}{.001359} = 2.06$$

  is approximately $\mathrm{N}(\theta, 1)$, where $\theta = (p_1 - p_2)/(.001359)$.
  Test $H_0 : \theta = 0$ versus $H_1 : \theta > 0$, based on z.
- Observed $z = 2.06$, so the p-value is 0.02.

Bayesian analysis:

Posterior odds of H_1 to H_0 = [Prior odds of H_1 to H_0] $\times B_{10}(z)$,

where

$$B_{10}(z) = \text{Bayes factor of } H_1 \text{ to } H_0 = \text{'data-based odds of } H_1 \text{ to } H_0\text{'}$$

$$= \frac{\text{average likelihood of H}_1}{\text{likelihood of H}_0 \text{ for observed data}} = \frac{\int \frac{1}{\sqrt{2\pi}} e^{-(z-\theta)^2/2} \pi(\theta) d\theta}{\frac{1}{\sqrt{2\pi}} e^{-(z-0)^2/2}},$$

For $z = 2.06$ and $\pi(\theta) = \text{Uniform}(0, 2.95)$, the nonincreasing prior *most favorable* to H_1,

$B_{10}(2.06) = 5.63$ (the p-value is 0.020, commonly misinterpreted as 50 : 1 odds)

(The actual subjective 'study team' prior yielded $B^*_{10}(2.06) = 4.0$, and the maximum Bayes factor over all possible priors is $e^{z^2/2} = 8.35$.)

Bayesian hypothesis testing and fixed α level testing are not compatible

- Fixed α level testing, i.e.
  - pre-experimentally choosing a rejection region $\mathcal{R}$ (e.g., $\mathcal{R} = (1.645, \infty)$ in the vaccine example) with Type I error probability $\alpha = Pr(\mathcal{R} \mid H_0)$,
  - and reporting the error as α, *no matter where the data is in* $\mathcal{R}$,

  is a valid frequentist procedure (as opposed to use of the p-value as the error probability, which is not a valid frequentist procedure).
- But it is an unconditional frequentist procedure and conditionally seems silly to a Bayesian, e.g. reporting the same $\alpha = 0.05$
  - when $z = 1.645$ (where 0.05 is a serious underestimate of the actual conditional error)
  - or $z = 5$ (where 0.05 is a serious overestimate of the actual conditional error).

Possible unification of frequentist and Bayesian testing

Setup (for now a mix of frequentist and Bayes):

We observe data $\boldsymbol{x}$ from the density $f(\boldsymbol{x} \mid \theta)$ and wish to test

$$H_0 : \theta = \theta_0 \quad (\text{or } H_0 : |\theta - \theta_0| < \epsilon) \quad \text{versus} \quad H_1 : \theta \neq \theta_0 \quad (\text{or } H_1 : |\theta - \theta_0| > \epsilon)\,.$$

- Suppose a rejection region $\mathcal{R}$ is specified.
- Let $\alpha = Pr(\mathcal{R} \mid \theta_0)$ and $(1 - \beta(\theta)) = Pr(\mathcal{R} \mid \theta)$ be the Type I error and power corresponding to the rejection region $\mathcal{R}$.
- Let π_0 and $\pi_1 = 1 - \pi_0$ be the prior probabilities of H_0 and H_1.
- Let $\pi(\theta)$ be the prior density of θ under H_1 (this could just be a point mass at a point θ' for which power is to be evaluated).
  - Then $(1 - \bar{\beta}) = \int (1 - \beta(\theta))\pi(\theta)d\theta$ is the average power wrt the prior $\pi(\theta)$ (equals $[1 - \beta(\theta')]$ if power at a point is used).
  - And $m(\boldsymbol{x}) = \int f(\boldsymbol{x} \mid \theta)\pi(\theta)d\theta$ is the marginal likelihood of the data $\boldsymbol{x}$ under the prior $\pi(\theta)$ (equals $f(\boldsymbol{x} \mid \theta')$ for a point mass prior).

Pre-experimental analysis (not new):

The pre-experimental probability of incorrectly rejecting H_0 is then $\pi_0\alpha$, while the pre-experimental probability of correctly rejecting H_0 is $\pi_1(1-\bar{\beta})$.

Definition: The *pre-experimental odds of correct to incorrect rejection of* H_0 are

$$\begin{aligned} O_{pre} &= \frac{\pi_1}{\pi_0} \times \frac{(1-\bar{\beta})}{\alpha} \\ &\equiv O_P \times R_{pre} \\ &\equiv [\text{prior odds of } H_1 \text{ to } H_0] \times [\textit{rejection odds} \text{ of } H_1 \text{ to } H_0]\,. \end{aligned}$$

Reporting of the rejection odds, R_{pre}, recognizes the crucial role of power in understanding the strength of evidence in rejecting, and does so in a simple way (reducing the evidence to a single number).

average power	0.05	0.25	0.50	0.75	1.0	0.01	0.25	0.50	0.75	1.0
type I error	0.05	0.05	0.05	0.05	0.05	0.01	0.01	0.01	0.01	0.01
R_{pre}	1	5	10	15	20	1	25	50	75	100

Example: Genome-wide Association Studies (GWAS)

- Early genomic epidemiological studies almost universally failed to replicate (estimates of the replication rate are as low as 1%), because they were doing extreme multiple testing at non-extreme p-values.
- A very influential paper in Nature (2007) by the Wellcome Trust Case Control Consortium proposed the cutoff $p < 5 \times 10^{-7}$.
  - Found 21 genome/disease associations; 20 have been replicated.
- The frequentist Bayesian argument for the cutoff:
  - They wanted an experiment with O_{pre}, the pre-experimental odds of a true to false positive, equal to $10:1$.
  - They assessed O_P, the prior odds of a true to false positive, to be $\frac{1}{100{,}000}$. (This is their implementation of Bayesian control for multiple testing; O_P could, instead, have been estimated from the data.)
  - Typical GWAS studies had power $(1-\bar{\beta}) = 0.5$.
  - Solving $[\frac{10}{1} = \frac{1}{100{,}000} \times \frac{0.5}{\alpha}]$ gave $\alpha = 5 \times 10^{-7}$.

Post-experimental odds analysis (not new):

Once the data is at hand a Bayesian would focus on the posterior odds of H_1 to H_0 given by

$$\begin{aligned} O_{post} &= \frac{\pi_1}{\pi_0} \times \frac{m(\boldsymbol{x})}{f(\boldsymbol{x} \mid \theta_0)} \\ &\equiv O_P \times R_{post}(\boldsymbol{x}) \,, \end{aligned}$$

where $R_{post}(\boldsymbol{x})$ is the data-dependent odds of a true to false rejection, more commonly called the Bayes factor of H_1 to H_0 and denoted $B_{10}(\boldsymbol{x})$.

GWAS example: Parts of the Nature article argued that it is best to just compute the Bayes factors, $B_{10}(\boldsymbol{x})$, and the posterior odds O_{post}.

For the 21 claimed associations, these ranged between

- $O_{post} = 10^{68}$ (overwhelming evidence of a correct rejection) and
- $O_{post} = \frac{1}{10}$ (evidence of an *incorrect* rejection; note that this is the one claimed association in the article that has *not* been replicated).

Reporting these these seems much more reasonable than always saying $O_{pre} = \frac{10}{1}$, but the article did not base decisions on them, presumably because they are not frequentist measures. Is that true?

Lemma (new): *The frequentist expectations of* $B_{10}(\boldsymbol{x})$ *and* $B_{01}(\boldsymbol{x}) = 1/B_{10}(\boldsymbol{x})$ *over the rejection region, conditional on the respective hypotheses, are*

$$E[B_{10}(\boldsymbol{x}) \mid H_0, \mathcal{R}] = R_{pre} = \frac{(1-\bar{\beta})}{\alpha}, \quad \textit{and} \quad E[B_{01}(\boldsymbol{x}) \mid H_1^*, \mathcal{R}] = [R_{pre}]^{-1},$$

where H_1^* *refers to the marginal alternative model with density* $m(\boldsymbol{x})$.

The first identity guarantees that, under H_0, the "average of the reported Bayes factors when rejecting" equals the actual rejection odds R_{pre}, so $B_{10}(\boldsymbol{x})$ *is as valid a frequentist report as is* R_{pre}.

How can a valid frequentist procedure depend on a prior distribution?

- Any power assessment requires at least specification of a point at which to assess power, and that can be used as the prior if nothing else is available.
- Thus, if one is willing to consider power, then R_{post} is much better than R_{pre}, since it has the same frequentist justification and is fully data dependent.

Vaccine Example:

There were widely varying opinions concerning the prior odds $O_p = \pi_1/\pi_0$, and the impact of this was illustrated and discussed in Gilbert et al., 2011.

But we will focus on O_{pre} and $O_{post}(z) = B_{10}(z)$, the odds arising from the experiment and data.

The observation is $z = 2.06$, where Z is approximately $\mathrm{N}(\theta, 1)$, and we are testing

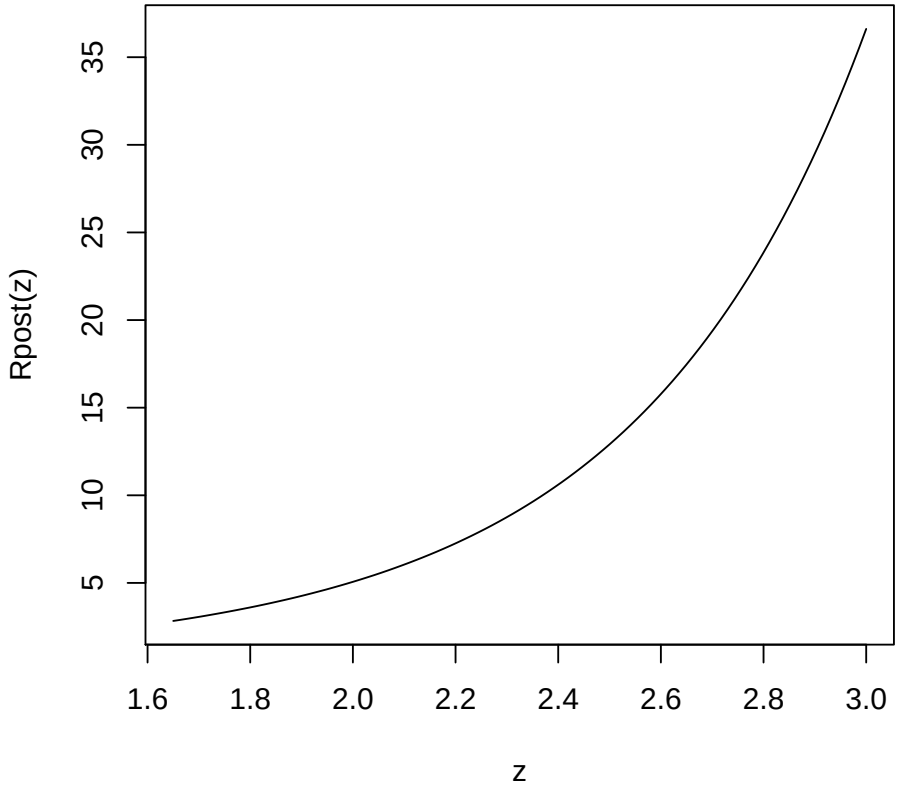
$$H_0 : \theta = 0 \quad \text{versus} \quad H_1 : \theta > 0\,.$$

The p-value is 0.02 and

$$O_{post}(z) = B_{10}(z) = \text{Bayes factor of } H_1 \text{ to } H_0 = \frac{\int \frac{1}{\sqrt{2\pi}} e^{-(z-\theta)^2/2} \pi(\theta) d\theta}{\frac{1}{\sqrt{2\pi}} e^{-(z-0)^2/2}}\,,$$

equaling $B_{10}(2.06) = 5.63$ for the actual data and using $\pi(\theta) = \text{Uniform}(0, 2.95)$, the nonincreasing prior *most favorable* to H_1.

Odds Analysis: $\mathcal{R} = (1.645, \infty)$, $\alpha = 0.05$ and the scientists said $1 - \bar{\beta} = 0.45$, so $R_{pre} = (1 - \bar{\beta})/\alpha = 9$. Thus, pre-experimentally, the rejection odds of a correct rejection to an incorrect rejection are nine to one.



Above is $B_{10}(z)$ (or $R_{post}(z)$), as a function of $z \in \mathcal{R}$. Thus, post-experimentally the odds of a correct rejection to incorrect rejection can be anywhere from 2 to ∞.

Recall $z = 2.06$ was observed, so $B_{10}(2.06) = 5.63$ is the actual odds.

Another argument for $B_{10}(\boldsymbol{x})$ as opposed to R_{pre}: (The basic issue pointed out to us by Deborah Mayo)

Pre-experimentally, the larger the power, the larger is R_{pre}, so the larger the probability that a rejection from the experiment will be correct, *assuming, of course, that the power has been accurately assessed.*

But the *opposite* behavior can be true post-experimentally, with fixed data.

Example: Suppose we use a point mass prior at θ' (corresponding to power at a point, and assume that this is indeed viewed as the likely alternative value).

- $R_{pre} = \frac{1-\beta(\theta')}{\alpha}$ will clearly be increasing for large θ'.
- $B_{10}(\boldsymbol{x}) = \frac{f(\boldsymbol{x}|\theta')}{f(\boldsymbol{x}|\theta_0)}$ will be decreasing for large enough θ' and fixed $\boldsymbol{x}$.

The intuition here is that the observation of (say) $p = 0.045$ can be *less likely* under a highly powered experiment (where much smaller p-values are expected if the alternative is true) than a moderately powered experiment, and so can be less evidence in favor of the alternative for the highly powered experiment. This is correctly reflected by $B_{10}(\boldsymbol{x})$, while R_{pre} incorrectly suggests the opposite.

A common complaint: determining Bayes factors is too hard. *But p-values can be converted into bounds on Bayes factors.*

Indeed, *robust Bayesian theory* suggests general and simple ways to calibrate p-values. (Vovk, 1993, Sellke, Bayarri and Berger, 2001, ELS, 1963).

Theorem 1 *A proper p-value satisfies $H_0 : p(X) \sim Uniform(0,1)$, so consider testing this versus $H_1 : p \sim g(p)$, where $Y = -\log(p)$ has a non-increasing failure rate (a natural non-parametric condition on g). Then*

$$B_{10} \leq \frac{1}{-e\, p\, \log(p)} \quad \text{for } p < e^{-1}.$$

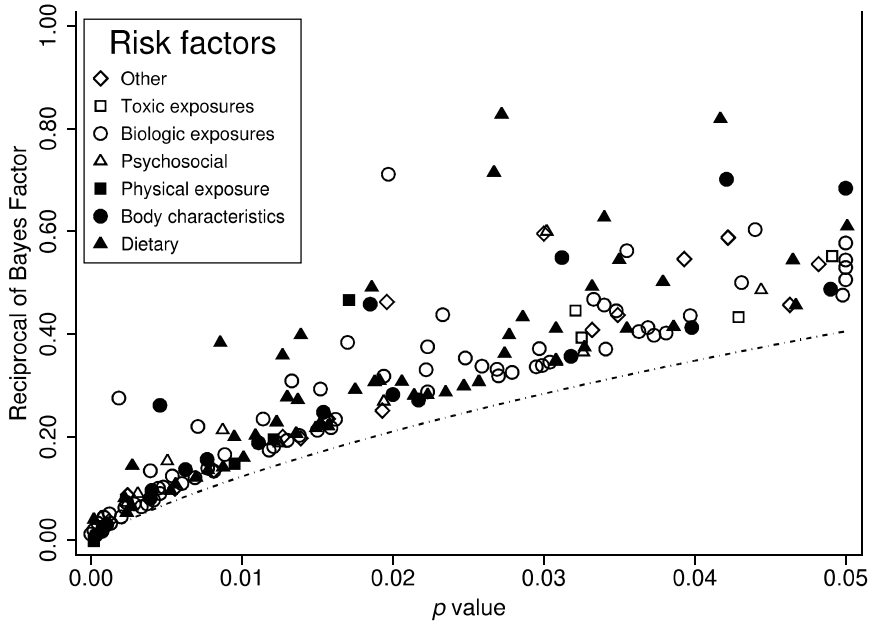
Theorem 2 *Consider testing $H_0 : \theta = \theta_0$ vs $H_1 : \theta \neq \theta_0$ based on test statistic $T(x)$, with $p(x) = P(T(X) > T(x) \mid \theta_0) \equiv 1 - F(T(x) \mid \theta_0)$ and f being the density corresponding to F. For* any *prior $\pi(\theta)$,*

$$B_{10} \leq \sup_\theta f(F^{-1}(1-p) \mid \theta) / f(F^{-1}(1-p) \mid \theta_0).$$

p	0.1	0.05	0.01	0.005	0.001	0.0001	0.00001	5×10^{-7}
$\frac{1}{-ep\log(p)}$	1.60	2.44	8.13	13.9	52.9	400	3226	2.0×10^5
$\sup_\theta$, Normal	2.9	4.4	14.7	25.7	113	970	8731	1.6×10^5

- Although very simple, there was initially concern that the $\frac{1}{-ep\log(p)}$ bound is too large, since it is known that Bayes factors can depend strongly on the sample size n, and the bounds do not.
- But the following studies indicate that this might not typically be a problem. These studies
  - look at large collections of published studies where $0 < p < 0.05$;
  - compute a Bayes factor, $B_{01} = 1/B_{10}$, for each study;
  - graph the Bayes factors versus the corresponding p-values.
- The lower boundary in all figures is essentially the lower bound $-e\,p\log(p)$ (the corresponding bound for $B_{01} = 1/B_{10}$ and given by the dashed lines in the figures), indicating that it is often an accurate bound.

The first two graphs are for 272 'significant' epidemiological studies with two different choices of the prior; the third for 50 'significant' meta-analyses (these three from J.P. Ioannides, Am J Epidemiology, 2008); and the last is for 314 ecological studies (reported in Elgersma and Green, 2011).


Risk factors
Other
Toxic exposures
Biologic exposures
Psychosocial
Physical exposure
Body characteristics
Dietary
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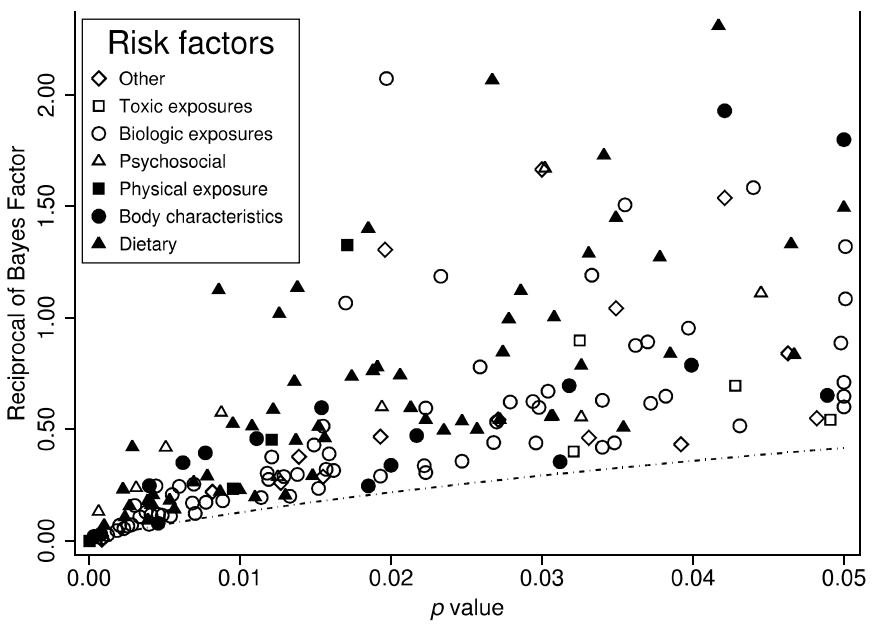
Risk factors
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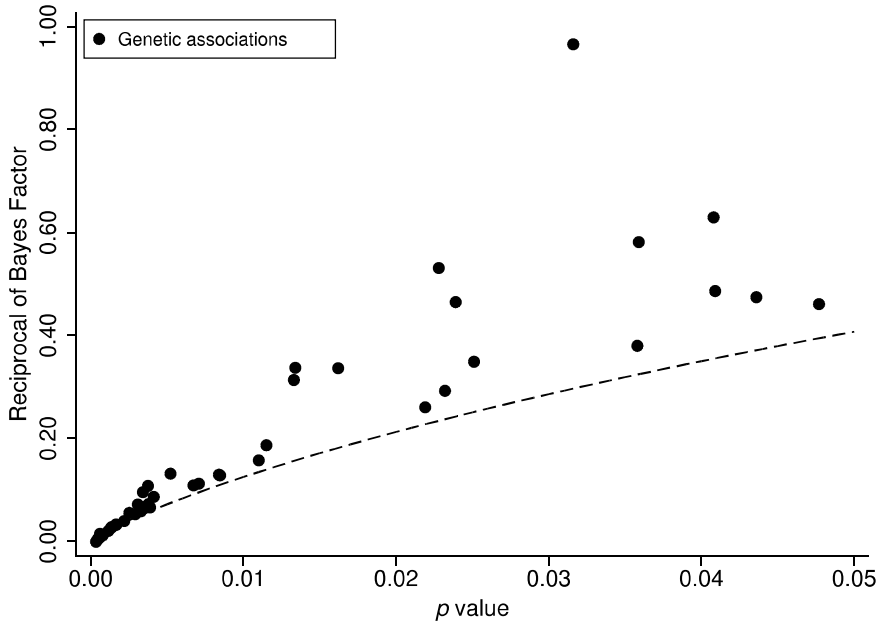
Genetic associations
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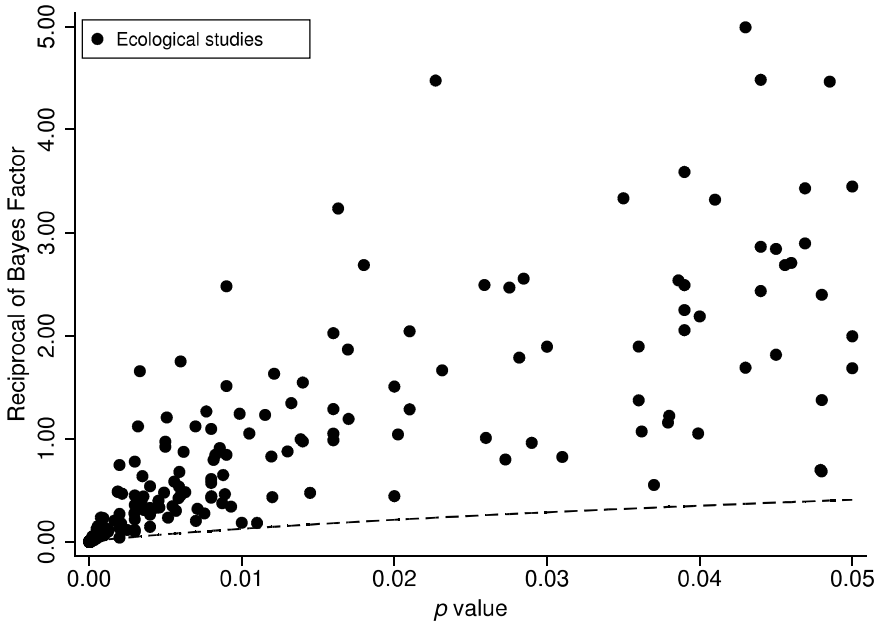
Ecological studies
Reciprocal of Bayes Factor
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p value

Bayesian and frequentist possibilities for choosing the prior $\pi(\theta)$ for the post-experimental rejection ratio or Bayes factor:

1. Subjective prior: When a subjective prior is available, such as the 'study team prior' in the vaccine example, using it is optimal. Again, note that the resulting procedure is still as much of a frequentist procedure as is the use of the pre-experimental rejection odds $O_{pre} = \frac{(1-\bar{\beta})}{\alpha}$.

2. Power considerations: If the experiment was designed with power considerations in mind, one can use the implicit prior that was utilized to determine power. This could be a prior distribution (or weight function) if used to compute power, or a specified point (i.e., a prior giving probability one to that point) if that is what was done.

3. Objective Bayes conventional priors: Discussion of these can be found in Berger and Pericchi (2001). One popular such prior, that applies to our testing problem, is the *intrinsic prior* defined as follows:

- Let $\pi^O(\theta)$ be a good estimation objective prior (using a constant prior will almost always work fine), with resulting posterior distribution and marginal distribution for data $\boldsymbol{x}$ given, respectively, by

$$\pi^O(\theta \mid \boldsymbol{x}) = f(\boldsymbol{x} \mid \theta)\pi^O(\theta)/m^O(\boldsymbol{x}), \quad m^O(\boldsymbol{x}) = \int f(\boldsymbol{x} \mid \theta)\pi^O(\theta)\, d\theta\,.$$

- Then the intrinsic prior (which will be proper) is

$$\pi^I(\theta) = \int \pi^O(\theta \mid \boldsymbol{x}^*) f(\boldsymbol{x}^* \mid \theta_0)\, d\boldsymbol{x}^*\,,$$

  with $\boldsymbol{x}^* = (x_1^*, \ldots, x_q^*)$ being imaginary data of the smallest sample size q such that $m^O(\boldsymbol{x}^*) < \infty$ (this is an imaginary bootstrap construction).

$\pi^I(\theta)$ is often available in closed form, but even if not, computation of the resulting Bayes factor is often a straightforward numerical exercise.

4. Empirical Bayes prior: This is found by maximizing the numerator of $R_{post}(\boldsymbol{x}) = B_{10}(\boldsymbol{x})$ over some class of possible priors. Common are the class of nonincreasing priors away from θ_0 (considered in the vaccine example) or even the class of all priors as in Theorem 2.

5. p-value bound: Instead of picking a prior distribution to calculate $R_{post}(\boldsymbol{x}) = B_{10}(\boldsymbol{x})$, use the generic upper bound $= B_{10}(\boldsymbol{x}) < 1/[-ep \log p]$.

These last two approaches have the problem that they are significantly biased (in the wrong way) from both Bayesian and frequentist perspectives. Indeed, if $\bar{R}_{post}(\boldsymbol{x})$ is the answer obtained from either approach, then

$$R_{post}(\boldsymbol{x}) < \bar{R}_{post}(\boldsymbol{x})\,, \qquad R_{pre} < E[\bar{R}_{post}(\boldsymbol{x}) \mid H_0, \mathcal{R}]\,.$$

Thus, in either case, one is reporting *larger* rejection ratios in favor of H_1 than is supported by the data (or experiment).
Of course, even though biased, use of either method would give much better answers than the usual misinterpretations of p-values.

Conclusion: saving the world from p-values

- We need to agree that the direct use of p-values as confirmatory evidence should stop (the ASA statement more or less says this); the historical evidence is clear that p-values cannot be properly interpreted by most users.
- The ideal replacement for p-values would be the posterior odds of H_1 to H_0:

$$O_{post} = O_P \times B_{10}(\boldsymbol{x}) \quad (\text{superior to } O_{pre} = O_P \times \tfrac{1-\bar{\beta}}{\alpha}),$$

  where O_P is the prior odds and $B_{10}(\boldsymbol{x})$ is the Bayes factor of H_1 to H_0.
- The Bayes factor can be the only report if use of prior odds is problematical. Possible choices of $\pi(\theta)$ for computing the Bayes factor:
  - A subjective prior or 'weight function' chosen during a pre-experimental power computation. (Note that $B_{10}(\boldsymbol{x})$ is as frequentist as $\frac{1-\bar{\beta}}{\alpha}$.)
  - A point mass at a value of θ used in a pre-experimental power calculation.
  - An objective prior distribution (e.g., the *intrinsic prior* for testing).
  - The prior from a class of priors that most favors H_1.
- If determination of $B_{10}(\boldsymbol{x})$ is not feasible, report the upper bound on the Bayes factor, $1/[-ep\log p]$; this is much less likely to be misinterpreted than p

Thanks!