Dogmatists Cannot Learn

To the Editor:

We wish to provide some rational in defense of the title of this comment. If we agree that a dogmatist is one whose beliefs cannot be influenced by observations (i.e., data), and we define learning as having your beliefs influenced by observations, then it follows that dogmatists cannot learn. While this statement has been made previously,[1(p.47)2] we believe it is useful to expand on this point with a simple example. Indeed, some dangers of dogmatism in epidemiology have been documented[3], and one does not have to look far for trivial examples of dogmatic statements.[4]

First, recall that the standard rules of probability theory are convexity, $0 \leq P(a) \leq 1$; multiplication, $P(a \text{ and } w) = P(a \mid w)P(w)$, where $P(a \mid w)$ is the probability of arbitrary statement a presuming arbitrary statement w holds; and addition, $P(a \text{ or } w) = P(a) + P(w) - P(a \text{ and } w)$. Next, we will need to review a simple application of probability theory which follows from the above rules,[5] and is adapted from an example given by Lindley.[6(p.131)] Here, we are using probability to denote belief, where probability 1 is certainty and probability 1/2 is equally likely as not. Although we also ascribe to probability as chance, and strive to follow Lewis' principal principle (i.e., to bring your belief probabilities in line with known chance-probabilities).[5]

Consider the following question: XDSWQGFCVDSUDZQVOQDQUYLLEMX? To make things interesting, this question is encrypted in a language we do not understand. For the purposes of this exercise, say we know that the possible answers to this question are 1/3 or 2/3. We will let θ denote this discrete parameter space, such that $\theta = 1/3$ or $\theta = 2/3$. We can extend to a continuous parameter space, but we wish to keep this example simple enough to tabulate.

Before we see any data, $n = 0$, we might have no knowledge about the relative probability of the two possible values of θ. To translate this ignorance into a prior probability, we employ Laplace's rule of succession,[7(p.19)] $P = (y+1)/(n+2)$, and assign probability 1/2 to each possible value of the statement. This means we have prior probability $P(\theta = 1/3) = P(\theta = 2/3) = 1/2$. To learn, we might gather observations or information. As an aside, information seems necessary but is not sufficient for learning. In addition to information, we require a system or engine to translate the information into knowledge. Here, in a setting where we wish to learn about the factual natural course (i.e., what is), we use probability logic as our engine.[8] In more complex settings where we wish to learn about what might be we use a counterfactual probability logic as our engine.[9] Information coupled with our engine and identification conditions appears sufficient for learning, but is not necessary (as other engines or conditions exist).

Say we find 12 people who can decrypt or translate this question and report to us whether they believe that $\theta = 1/3$ or $\theta = 2/3$. For simplicity, we will consider these 12 people independent and exchangeable[10] (or permutable[11]) with respect to their decryption abilities. Also, we will ignore the certainty with which their reports are provided. Say the reports are 7 of 12 in favor of $\theta = 2/3$. Recall that Bayes' rule is $P(w|a) = P(a|w)P(w)/P(a)$. In our setting, this is $P(\theta|y) = P(y|\theta)P(\theta)/P(y)$, where y is shorthand for the observed data y/n and $P(y|\theta)$ is the likelihood.[12] In our setting, the likelihood is binomial, or $P(y \mid \theta) = \binom{n}{y}\theta^{y}(1-\theta)^{n-y}$. Details of calculations are shown in the upper panel of the Table. Our posterior probability that $\theta = 2/3$ is 0.8, much increased from our prior probability that $\theta = 2/3$, which was 0.5.

Now we are prepared to demonstrate that dogmatists cannot learn. Say our prior probabilities are as given in the lower panel of the Table. This dogmatic prior probability completely rules out the option $\theta = 2/3$. The data and resultant likelihood are unchanged from the upper panel of the Table. Regardless of

TABLE. Posterior Probability Distributions Under Two Different Priors

θ	Prior Probability $P(\theta)$	Likelihood $P(y \mid \theta)^a$	$P(y \mid \theta)P(\theta)$	Posterior Probability $P(\theta \mid y)^b$
Laplace Prior				
1/3	0.5	0.04769	0.02385	0.2
2/3	0.5	0.19076	0.09538	0.8
Total	1.0	—	$P(y)$=0.11922[c]	1.0
Dogmatic Prior				
1/3	1.0	0.04769	0.04769	1.0
2/3	0.0	0.19076	0.0	0.0
Total	1.0	—	$P(y)$=0.04769[c]	1.0

[a] $P(y \mid \theta) = \binom{n}{y}\theta^{y}(1-\theta)^{n-y}$.

[b] $P(\theta \mid y) = P(y \mid \theta)P(\theta)/P(y)$.

[c] $P(y) = \Sigma_{\theta} P(y \mid \theta)P(\theta)$.

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the data, here the dogmatists' posterior equals their prior. Therefore, dogmatists cannot learn. Of course, this prior statement (and the title of this comment) is itself dogmatic, although we encourage dissent.[2,13] Finally, we are usually dogmatic regarding options we do not foresee. A point of this comment is to prepare ourselves to be open to alternatives as they present themselves. As Wittgenstein said: "What is thinkable is also possible."[14(comment 3.02)]

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REFERENCES

1. Savage LJ. *The Foundations of Statistics*. 2nd ed. New York, NY: Dover; 1972.
2. Cole SR. Nondogmatism. *Ann Epidemiol*. 2016;26:231–233.
3. Stolley PD. When genius errs: R.A. Fisher and the lung cancer controversy. *Am J Epidemiol*. 1991;133:416–425; discussion 426.
4. Cole SR. Atheoretical science: a response to the poverty of popperian epidemiology. *Int J Epidemiol*. 1996;25:899–900.
5. Greenland S. Probability logic and probabilistic induction. *Epidemiology*. 1998;9:322–332.
6. Lindley DV. *Understanding Uncertainty*. Revised ed. New York, NY: John Wiley & Sons; 2014.
7. Laplace PS. *A Philosophical Essay on Probabilities (Translated in 1902 by Truscott, F.W., Emory, F. W.)*. New York, NY: Wiley; 1814.
8. Jaynes ET. *Probability Theory: The Logic of Science*. Cambridge, UK: Cambridge University Press; 2003.
9. Robins JM. A new approach to causal inference in mortality studies with a sustained exposure period: application to control of the healthy worker survivor effect. *Math Model*. 1986;7:1393–1512.
10. de Finetti B. Foresight: Its logical laws, its subjective sources. *Annales de l'Institu Henri Poincare*. 1937;7:95–157.
11. Good IJ. *Good Thinking*. Minneapolis, MN: University of Minneapolis Press; 1983.
12. Cole SR, Chu H, Greenland S. Maximum likelihood, profile likelihood, and penalized likelihood: a primer. *Am J Epidemiol*. 2014;179:252–260.
13. Watkins JWN. Comprehensively critical rationalism. *Philosophy*. 1969;44:57–62.
14. Wittgenstein L. *Tractatus Logico-Philosophicus*. New York, NY: Harcourt, Brace & Company; 1921.

Using the Lorenz Curve to Assess the Feasibility of Targeted Screening for Esophageal Adenocarcinoma

To the Editor:

The incidence of esophageal adenocarcinoma has been rapidly increasing in the Western societies in recent decades.[1] Gastroesophageal reflux disease, obesity, and tobacco smoking are the major risk factors.[1,2] Esophageal adenocarcinoma is characterized by a poor prognosis with an overall 5-year survival below 15%–20%, which is even worse in patients diagnosed at late stages.[1] Upper endoscopy is increasingly utilized for detection of the premalignant condition of esophageal adenocarcinoma, that is, Barrett's esophagus with dysplasia. However, a universal screening strategy, even in patients with reflux, is unfeasible given the considerable costs and risk of complications, and the low incidence (0.7 per 100,000 person-years globally, and the highest, 7 per 100,000 years in men in the United Kingdom).[3,4] Risk prediction models for esophageal adenocarcinoma combining information on risk factors have recently been developed. These have had good discriminative accuracy and have shown promising potential in identifying high-risk individuals who might benefit from targeted prevention and early detection strategies.[5,6]

In this study, we further assessed the feasibility of targeted screening based on a risk prediction model using the Lorenz Curve. The Lorenz Curve is a graphical tool widely used in econometrics to characterizing the distribution of wealth in the society, which has been suggested to be valuable in demonstrating the "concentration" of disease risks.[7] This tool may be particularly relevant when evaluating screening programs in the context of disease risk prediction.

We have developed a risk prediction model based on data from a nationwide population-based case–control study in Sweden in 1995–1997.[2,5] Participants included 189 histologically confirmed incident cases of esophageal adenocarcinoma from all relevant hospital departments in Sweden, and 820 control subjects frequency-matched for age and sex and randomly selected from the Swedish population. Detailed information on risk factors was collected via face-to-face interviews. This study was approved by all six regional ethical review boards in Sweden, and both written and oral informed consent was obtained from each participant. In brief, we ranked the estimated risks for all controls (representing the population at risk) obtained from the logistic regression model. For each of the risk levels, the cumulative proportions of controls and estimated risks having this level of risk or below were used to draw the curve. Methodological details can be found in the report by Mauguen and Begg.[7]

The constructed Lorenz Curve is shown in Figure. Based on a simple model, which only included information on reflux symptoms or use of antireflux medication, body mass index, and tobacco smoking, 37% of all esophageal adenocarcinoma cases would occur in the 10% of the population with the highest risks, and 22% of all cases would be identifiable from the top 5% of the population based on risk. The estimated risks would be even more concentrated after also considering age and sex. For example, the risk concentration should

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