who will not make themselves available unless they think their expertise will count for something. It is said that laws are like sausages: seeing what goes into them destroys one's confidence in them. Scientific judgments made on behalf of the public must not come to that.

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Lyme Disease Study

Eliot Marshall's News & Comment article (13 Oct., p. 228) concerning the National Institutes of Health's (NIH's) study to settle the dispute concerning Lyme disease was a relief to many on both sides of this controversy. To ensure that this debate is settled fairly and convincingly, both sides should be fully represented and deeply involved in the planning and design of the study, and both should agree formally about the protocol—before the results are in. Both sides should agree on what specific results would support which side of the debated issue. Statistics should be involved in the design, analysis, and interpretation, and at least one of them should be outside the medical community.

The analysis should not be based on only the standard results of hypothesis tests and their associated problems (for example, the arbitrary α level, the multiple testing problem, or assumed asymptotic distribution of the test statistic). I suggest the use of an informational theoretic approach to inference, such as Akaike's information criterion, in addition to the traditional testing approach. With these caveats, I hope the study is funded and conducted.

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Glucocorticoids

Jean Marx describes the discovery of interaction between the transcription factor NF-κB and the glucocorticoid receptor in her Research News article "How the glucocorticoids suppress immunity" (13 Oct., p. 232). Readers may incorrectly conclude that this observation was simultaneously made by four laboratories a year ago. Our description (*1*) of a direct interaction between NF-κB and the glucocorticoid receptor, which established NF-κB as a target in anti-inflammation, was published almost 2 years ago. A year later, Scheinman *et al.* and Caldenhoven *et al.* reported similar data (*2*). We believe that existing evidence does not allow a generalized assumption that upregulation of IκBα production is *the* major pathway by which glucocorticoids repress NF-κB–mediated activation of all target genes by all stimuli. An interaction between NF-κB and the glucocorticoid receptor appears to play a significant role in the rapid and efficient transcriptional repression of the inflammation-associated cytokine interleukin-6 gene by glucocorticoids.

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