and the results of the meta-analysis less relevant in the context of the hypothesis that ongoing treatment with azithromycin would lead to sudden death from cardiovascular causes, as suggested by the study involving Medicaid beneficiaries by Ray et al.

Brass argues that our conclusion is not supported by our results. Our conclusion was carefully phrased with the use of noncausal terminology and is an accurate reflection of our findings that exposure was not associated with an increased risk of the outcome. This reflects a statistical estimate that best fits the data, given the imposed statistical model.

However, like Brass, we think it is important to note that only a moderate-to-high relative increase in the risk of death from cardiovascular causes (>55%) can be ruled out with a high degree of certainty; this was explicitly stated in the Discussion section of our article.

Nevertheless, Brass raises important questions about terminology and the interpretation of statistical measures in medical research. We believe these questions extend far beyond the context of our specific study.

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Since publication of their article, the authors report no further potential conflict of interest.

DOI: 10.1056/NEJMc1306999

DR. MOSHOLDER AND A COLLEAGUE REPLY: McMurray and Jhund correctly point out that mortality was not increased in long-term placebo-controlled trials of azithromycin for the prevention of atherosclerotic cardiac events. However, we draw only very limited reassurance from these data because of the design of the trials. In the six trials analyzed by Baker and Couch, the duration of azithromycin exposure relative to the follow-up time was different from that in the study by Ray et al., because patients received azithromycin only during a small fraction of the total days of follow-up time. For example, in the Azithromycin and Coronary Events Study reported by Grayston et al., patients received azithromycin once a week for a year, and the median time to follow-up was 3.9 years. Given that Ray et al. found a risk only on days with azithromycin use (days 1 through 5 after the prescription), these long-term trials on cardiovascular prevention, as they were analyzed, are not suitable for assessing such an association with the rate of death from cardiovascular causes.

The comments by Brass reinforce the importance of considering not only the point estimate for a measure of risk, but also its confidence interval.

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DOI: 10.1056/NEJMc1306999

Effects of Medicaid on Clinical Outcomes

TO THE EDITOR: In a comprehensive and careful follow-up to their previous analysis,[1] Baicker et al. (May 2 issue)[2] report on the effects of insurance coverage on health care and health outcomes in the Oregon Medicaid lottery experiment after approximately 2 years. Their instrumental-variable analysis is the next best thing to a randomized, controlled trial, since the instrument — in this case, winning a lottery for Medicaid coverage — satisfies the large-sample properties of being correlated to treatment and not being correlated to the outcomes of interest (e.g., health care utilization and outcomes) except through its effect on treatment.[3]

The financial effects on the lottery winners were not trivial. They received an in-kind benefit valued at one third to two thirds of their household income, their out-of-pocket spending was reduced by 39% ($215), and catastrophic expenditures were reduced by 81%. These financial consequences could have a direct effect on self-reported depression, other mental health conditions, and possibly other outcomes. It is telling that self-reported happiness increased in the first year after winning the lottery, but not in the second.[1,2,4] Awarding lottery winners equivalents of cash prizes (worth approximately $6,600 each) rather than Medicaid might have

improved their health outcomes and well-being even more.

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No potential conflict of interest relevant to this letter was reported.

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DOI: 10.1056/NEJMc1306867

TO THE EDITOR: The abstract in the article by Baicker et al. states that "Medicaid coverage generated no significant improvements in measured physical health." This is a misleading summary of the data reported in their article. The best estimates are that the Medicaid group had better outcomes than the control group according to most measures (see Table 2 of the article). The problem is that these findings are not statistically significant.

So, the effects might have been zero. That is not the same as saying that they were zero, or even that they were small. Buried toward the end of the article is the statement, "The 95% confidence intervals for many of the estimates of effects . . . include changes that would be considered clinically significant."

Nevertheless, almost all the article, the related editorial,[1] and related news reports, opinion pieces, and online discussions proceeded as if the effects had been found to be zero.

If one objects, on the basis of a lack of statistical certainty, to the simple summary that the Medicaid group had better outcomes, then one should describe the substantive meaning of the confidence interval. An honest summary is that it is quite likely there were positive effects, though it is possible that they were zero or negative.

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No potential conflict of interest relevant to this letter was reported.

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DOI: 10.1056/NEJMc1306867

TO THE EDITOR: Baicker et al. provide high-quality evidence of the failure of insurance to promote physical health even among the insured. One cause may be that insurers do not cover cost-effective programs that reach beyond the threshold of the physician's office to change patient behavior,[1] support patients at home,[2] and alter systems of delivery.[3]

Even if they suggest that they support such interventions, insurers underinvest in them. For instance, for persons with diabetes, my own (generous) insurance covers only a handful of visits with a dietitian, and it offers neither telephone-based reminders nor counseling on diet or exercise — far less than effective methods require.[4]

Public and private payers have tried to compensate hospitals and physicians according to performance. Results have been decidedly mixed.[5] The reason may be not what they pay or how they pay it, but to whom. Insurers should be rewarded and penalized on the basis of comprehensive health outcomes (e.g., mortality, not just blood-pressure levels).

Insurers determine where funds flow. If we want the health system to care more about population health, we should make sure that insurers do too.

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No potential conflict of interest relevant to this letter was reported.

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DOI: 10.1056/NEJMc1306867

THE AUTHORS REPLY: We appreciate the attention given to our study and agree with many of the comments. Our study indeed does not speak directly to the effects of different types of insurance or of alternatives such as cash grants; these are important topics for future research.

Statistical precision is crucial to interpretation of the findings. The reported confidence intervals do not allow us to reject the null hypothesis that there was no effect of Medicaid on blood-pressure, cholesterol, or glycated hemoglobin levels — but they are also consistent with Medicaid improving (or harming) these outcomes. Empirical estimates always come with uncertainty. A key question is what effect sizes our findings rule out, and how these compare with findings from previous studies of the effect of health insurance or with expectations based on available treatments.

In some cases, we can reject effect sizes seen in previous studies. For example, we can reject decreases in diastolic blood pressure of more than 2.7 mm Hg (or 3.2 mm Hg in patients with a preexisting diagnosis of hypertension) with 95% confidence. Quasi-experimental studies of the 1-year effect of Medicaid showed decreases in diastolic blood pressure of 6 to 9 mm Hg.[1,2]

In other cases, our confidence intervals do not rule out the health improvements one might expect given our estimate of the effect of Medicaid on medication use. For example, as noted in our article, given our estimate of the increase in the use of diabetes medication because of Medicaid, the clinical literature would predict a decrease in the average glycated hemoglobin level of 0.05 percentage points, an effect that is well within our 95% confidence interval.

We assessed conditions for which treatments exist that were effective within 2 years (our study period), but power is always constrained by sample size and is further reduced here by the imperfect take-up rates of Medicaid and lack of baseline clinical measures. We took several steps to increase power. Our study examined subgroups in which one might expect larger Medicaid effects (older persons and those with preexisting conditions) and we combined measures using the composite Framingham risk score. In none of these cases did we find statistically significant effects of Medicaid on physical health. We did find substantial and significant improvements in depression and financial well-being, as well as an increased use of health care.

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DOI: 10.1056/NEJMc1306867

Case 12-2013: A Woman with Pulmonary Infiltrates and Respiratory Failure

TO THE EDITOR: The Case Record presented by Hunt et al. (April 18 issue)[1] describes a previously healthy 18-year-old woman with severe pneumonia due to herpes simplex virus type 1 (HSV-1). It was speculated that the infection may have been acquired from her new boyfriend, and the HSV-1 infection was more likely to be primary than due to reactivation. The large number of infected cells in the bronchoalveolar-lavage fluid, the appearance of the alveolar cells on cytologic examination, and the rapid response to therapy with acyclovir were cited as reasons for the diagnosis of probable primary HSV infection. Determination of the patient's HSV IgG and IgM status would have further helped address this issue. Although serum antibody against HSV-1 was positive, the type of antibody was not mentioned. A positive IgM (with negative IgG) would suggest primary infection, whereas a positive IgG (with negative IgM) would indicate HSV reactivation, either of which may have led to pneumonia and the crusted lesion on the patient's lip. Also, clar-