(HAPO) study.[3,4] Fifteen institutional review boards approved our protocol, and none rejected the trial on ethical grounds. In addition, an independent data and safety monitoring committee regularly reviewed the trial. Some participants would not even have met the criteria for gestational diabetes (more evidence of clinical equipoise). We strongly disagree with an assertion that we unnecessarily exposed participants to harm. Randomized trials are necessary provided that there is sufficient uncertainty and that the results will resolve the dispute among physicians, which was the case for the treatment of mild gestational diabetes.[5] We agree that our results may encourage previously reluctant physicians to treat gestational diabetes, but such treatment is now supported by evidence from a clinical trial rather than by expert opinion alone.

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

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Dectin-1 Deficiency and Mucocutaneous Fungal Infections

TO THE EDITOR: Mucocutaneous candidiasis is commonly seen in persons with deficient T-cell–mediated immunity, including newborn infants, patients with the acquired immunodeficiency syndrome, and those with genetically defined primary T-cell deficiencies.[1] In their Brief Report about human dectin-1 deficiency and mucocutaneous fungal infections, Ferwerda et al. (Oct. 29 issue)[2] suggest that chronic mucocutaneous candidiasis may also be caused by a genetic defect of the β-glucan receptor dectin-1. However, their data should be interpreted with caution. First, they provide no information about how dectin-1 deficiency impairs overall T-cell–dependent mucosal immunity against candida. Second, they suggest that the lack of invasive fungal infection in dectin-1–deficient patients may be attributed to the normal phagocytosis and killing of candida by granulocytes. This explanation sounds reasonable but conflicts with observations of invasive microbial disease in patients with other, recently described innate immune deficiencies.[3,4] Finally, it is difficult to define the precise immunologic role of dectin-1 in immunity to candida in light of the demonstration by Saijo et al.[5] that dectin-1 may not actually be required for host defense against *Candida albicans*.

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THE AUTHORS REPLY: In response to Maródi and Erdös: T-cell–derived immunity, and especially responses of type 17 helper T cells (Th17), are crucial for mucosal antifungal defense.[1,2] Our report describes deficient Th17-cell responses in patients with dectin-1 deficiency,[3] and a similar defect has been reported in persons with defective

CARD9, the caspase recruitment domain–containing protein 9.[3] The fact that dectin-1 deficiency increases susceptibility to mucocutaneous, but not systemic, fungal infections stresses the specific role that dectin-1 may have in mucosal antifungal host defense. This is not in conflict with a role of NEMO (the inhibitor of kappa light polypeptide gene enhancer in B cells, kinase gamma), IRAK-4 (the interleukin-1 receptor–associated kinase 4), or MyD88 (the protein encoded by myeloid differentiation primary response gene 88) for the host defense against systemic bacterial or fungal infections, since these are adaptor molecules specific for toll-like receptors, a different class of pattern-recognition receptors.[4] The suggestion by Saijo et al.[5] in their study that dectin-1 is not involved in the host defense against systemic candidiasis in mice reinforces the conclusion that dectin-1 may be specific for mucosal antifungal defense.

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

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Taxing Sugar-Sweetened Beverages

TO THE EDITOR: Brownell et al. (Oct. 15 issue)[1] provide support for a tax on sugar-sweetened beverages in part by citing the results of long-term, randomized, controlled trials. They cite a report of a 1-year trial involving students 7 to 11 years of age that showed a lower incidence of obesity in the dietary intervention group, although the difference in body-mass index was not significant.[2] Follow-up 2 years after completion of the trial showed that the difference in the incidence of obesity was not sustained.[3] This dietary intervention apparently only had a transient effect without affecting the long-term propensity for obesity. None of the three other long-term, randomized, controlled trials cited in the article met their primary end points; an analysis of a different subgroup within each trial was made in an attempt to show some benefit.

The essential failure of these trials should give us pause. Before assigning blame for the obesity epidemic, we should have clinical evidence that an intervention to reduce the consumption of sugar-sweetened beverages is effective in achieving this goal, is either more effective or additive to the effect of other proven dietary therapies, and will reduce the long-term propensity for obesity.

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

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TO THE EDITOR: Although their intent is unquestionably noble, Brownell and coauthors do not produce an economic rationale for a soda tax; they merely present one side of the economic equation (cost–harm) without consideration of its usefulness. To maintain liberty, we defer to individual persons to balance the cost–utility equation. However, the authors point to market failure as a justification for intervention. Their first two rationales involve the argument that the population lacks the capacity to make free economic decisions. In attempting to restrict peoples' liberty, the onus is on the authors to convincingly