

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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2. James J, Thomas P, Cavan D, Kerr D. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *BMJ* 2004;328:1237.
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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

show that the vast majority of the population has no concept that consuming junk food (including soda) in excess has the potential for adverse health effects. Yet, their article includes data showing that a majority of people support a tax on health grounds. Their final rationale involves health care expenditure. It is a legitimate political issue, but not an economic justification to override autonomy. If instituted for “health” reasons, such a tax would be the ethical equivalent of compulsory participation in a program for the sake of the greater good; this is an anathema to modern ethics.

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TO THE EDITOR: Brownell et al. present a convincing argument for a link between the consumption of sugar-sweetened beverages and obesity and for the taxation of such beverages from a public health standpoint. Little discussion is given to the issue of consistency in the implementation

of such a tax policy. Many behavioral choices involve costs borne by society. All high-caloric foods can be tied to obesity. If soda is taxed, should this tax also be applied to all “fast food,” confections, or portion size? Why limit it to food? Should we not tax all behaviors linked to health care expenditures? Why not deter gun and motorcycle ownership or sedentary lifestyle through taxation? How parental should government be?

Government clearly has an important role in promoting public health, but singling out sugar-sweetened beverages may appear arbitrary. The authors cite favorable public opinion toward such a tax. How might public opinion change if the proposed tax was on all “unhealthy” personal choices? Where do the authors believe the line should be drawn? Which behaviors warrant taxation, and which should be excluded from taxation?

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

Somatic Mutations of *IDH1* and *IDH2* in the Leukemic Transformation of Myeloproliferative Neoplasms

TO THE EDITOR: Somatic mutations affecting the R132 residue of isocitrate dehydrogenase 1 (*IDH1*) and the homologous *IDH2* R172 occur in central nervous system tumors.^{1,2} Recently (in the Sept. 10 issue of the *Journal*³), alterations of *IDH1* R132 (in exon 2) were reported in 16 of 188 patients with de novo acute myeloid leukemia, with a strong association with a normal karyotype; however,

mutations of *IDH2* R172 (in exon 4) were not detected. We sequenced exon 2 of the *IDH1* gene and exon 4 of the *IDH2* gene in patients with leukemia that had evolved from a myeloproliferative neoplasm harboring a mutation in the Janus kinase 2 (*JAK2*) gene. Somatic mutations in *IDH1* or *IDH2* were present in 5 of 16 patients (31%) (Table 1), but these mutations were not present in

Table 1. *IDH* Mutations at the Leukemic Transformation of a Myeloproliferative Neoplasm with a *JAK2* Mutation in Five Patients.*

Patient No.	Preceding Myeloproliferative Neoplasm		At Leukemic Transformation		
	Phenotype	<i>JAK2</i> Mutation	<i>JAK2</i> Status	Karyotype	<i>IDH</i> Mutation
1	Primary myelofibrosis	V617F	Mutant	Not done	<i>IDH1</i> R132C
2	Primary myelofibrosis	V617F	Mutant	del(7q), +8	<i>IDH2</i> R140Q
3	Polycythemia vera	V617F	Wild type	Complex	<i>IDH2</i> R140Q
4	Essential thrombocythemia	V617F	Wild type	Not done	<i>IDH1</i> R132C
5	Polycythemia vera	Exon 12	Wild type	Normal	<i>IDH1</i> R132C

* “Not done” indicates that cytogenetic analysis was not performed, and +8 denotes trisomy 8.