

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

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

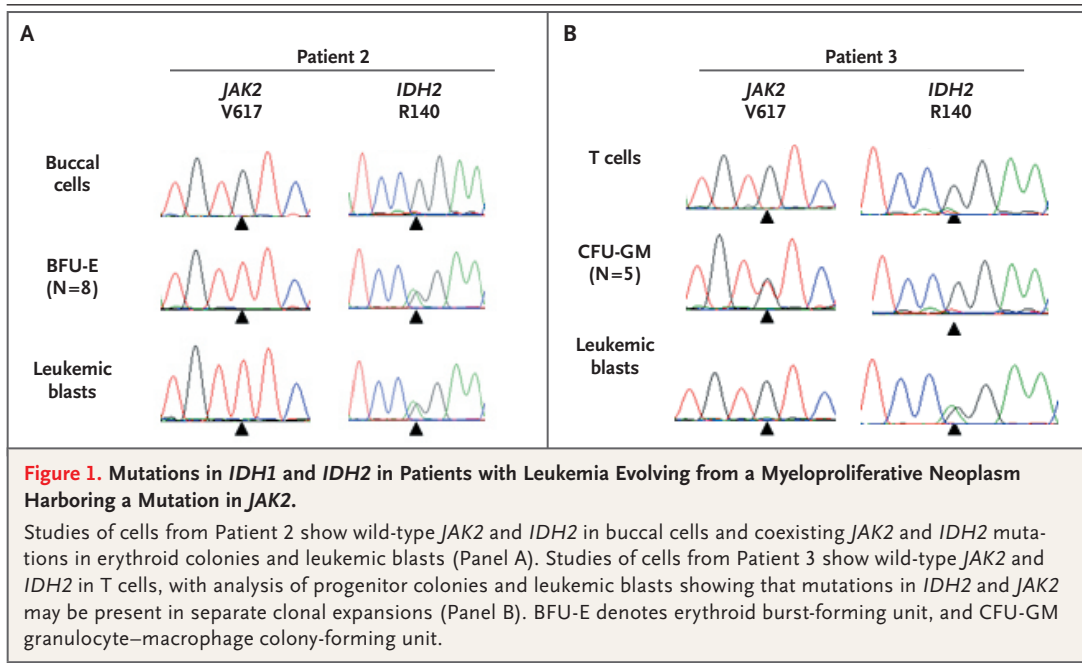
TO THE EDITOR: Somatic mutations affecting the R132 residue of isocitrate dehydrogenase 1 (*IDH1*) and the homologous *IDH2* R172 occur in central nervous system tumors.^{1,2} Recently (in the Sept. 10 issue of the *Journal*³), alterations of *IDH1* R132 (in exon 2) were reported in 16 of 188 patients 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.



180 unselected patients with chronic-phase polycythemia vera or essential thrombocythemia. Among the five patients with *IDH* mutations, three harbored an *IDH1* R132C substitution and two harbored a novel *IDH2* R140Q mutation affecting a residue conserved in mouse, yeast, and plant homologues.

In Patient 2, an *IDH2* R140Q mutation was detected in erythroid colonies with a *JAK2* mutation as well as in leukemic blasts (Fig. 1A), indicating that this alteration was acquired early during the progression to leukemia and is not responsible for the block in cellular differentiation that occurs in acute leukemia. Leukemic cells arising from myeloproliferative neoplasms with a *JAK2* mutation are commonly *JAK2* wild-type,⁴ although the mechanism by which they become wild-type is unknown. Among the five samples of leukemia cells carrying an *IDH* mutation, three samples lacked the *JAK2* mutation. In two of these three samples, an *IDH* mutation was present in leukemic blasts with wild-type *JAK2* but absent from progenitor colonies with a *JAK2* mutation (Fig. 1B), establishing the presence of two sepa-

rate clonal expansions in each patient. In these cases, the separate *JAK2*-mutant and *IDH*-mutant clones may represent the shared progeny of an ancestral abnormal clone or the coexistence of two distinct disorders with independent origins.⁵

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Responses to 2009 H1N1 Vaccine in Children 3 to 17 Years of Age

TO THE EDITOR: The current 2009 pandemic influenza A (H1N1) virus is associated with substantial morbidity in children, with 45% of hos-

pitalizations occurring in patients under 18 years of age.¹ One possible reason for this trend is a lack of preexisting immunity against the 2009