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The Importance of Understanding the Impact of Children's Food Marketing on the Brain

A central component of understanding the impact of the food environment on the obesity epidemic will be to explore the influence of industry-developed cues on the brain and behavior. Understanding the contribution of advertising to problematic eating is essential, especially because this may inform potential policy approaches to improve children's health and welfare. Children currently view approximately 6000 commercials annually and, despite self-regulation by the industry, the majority of these advertisements feature calorie-dense and nutrient-poor foods.[1]

See related article, p 759

Advertising ("commercial speech") has been granted protection by the courts under the First Amendment, even though considerable harm might accrue.[2] There are some examples of restrictions in marketing to children, when the products are illegal to sell to children, (eg, cigarettes and alcohol[3]), but food marketing to children seems as lawless as the wild west. Any food can be marketed in any way, to any age group, and even the most vulnerable demographic groups can be targeted.

Attention to the food marketing issue, and hence calls for regulation, is likely to increase as the public and elected leaders become aware of the social cost, particularly with respect to vulnerable groups such as children. The case against marketing may become especially compelling as its neurobiologic effects are better understood. Parents and public officials may not be happy knowing the impact of marketing on children's brains and may be even more concerned to find that companies are doing neuroscience research themselves, presumably to fully exploit this potential.

In this issue of *The Journal*, Bruce et al[4] lay important groundwork for continued exploration into how branded food items featured may impact neurobiology and affect childhood obesity. One of the main findings was the hypoactivation of control-related neural regions (ie, bilateral middle/inferior prefrontal cortex) to food logos in obese compared with lean children.

It will be tempting to suggest interventions to assist at-risk or obese children in the development of enhanced control strategies to help them resist marketed foods. This may have some utility, but who would pay for and disseminate such an approach to the extent large numbers of people would benefit? In addition, the food industry could readily undo any such effort by simply advertising more and more effectively.

Why not simply restrict the toxic influence? Policies that place restrictions on the marketing of calorie-dense, nutrient-poor foods (eg, sugar-sweetened beverages) to children will be comparatively inexpensive, more rapidly and broadly effective, and will actively reduce the environmental stimuli that may contribute to diminished control over eating compared with approaches that accept the presence of marketing and rely on children to resist.

Another important component to environmentally focused policies is the ability to encourage healthier behavior across a broader spectrum of the population. Intervention approaches often target smaller clinical subgroups or at-risk individuals. These subgroups are important targets for additional assistance, but the public health impact of the negative food environment challenges the ability of the majority of individuals to maintain a healthy relationship with food. Bruce et al[4] found that exposure just to food logos in lean children generated greater activation in regions that have been related to future weight gain and difficulty with weight maintenance in response to food cues (eg, middle frontal gyrus, middle temporal gyrus, insula).[5] Thus, activation in response to food logos may predict future weight gain in children who are not currently obese. Further, lean children may be using greater control strategies to reduce their response to food logos, but the ubiquitous nature of food advertisements could potentially deplete these resources because of repeated exposure.[6] These possibilities highlight the importance of improving the food environment, not only for obese children, but also for normal-weight children.

The research into food marketing also leads to the consideration of policy approaches related to minimizing brand exposure not only from traditional media of branded content (eg, television commercials) but also the more novel uses of logos by the food industry. Food brands are already commonplace in a number of arenas that are frequented by children (eg, sporting facilities, schools), but more aggressive campaigns also have started to use

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branding in online advergaming, in school buses, on speed-bumps, and in social media.[7] The increased exposure to branding through these means might result in greater behavioral and biological response to food logos in future generations. Further, minorities often are targeted for increased marketing of unhealthy foods, especially African-American and Hispanic youth. Targeted advertising has been related to greater consumption of high-calorie foods (eg, fast foods) by African-American and Hispanic children.[8] This trend might also be related to greater neural response to food logos in children from racial/ethnic groups at increased risk of obesity, which might highlight the need for policy to restrict the targeted food marketing to minority children.

In summary, the research conducted by Bruce et al[4] helps lay essential groundwork for an important area of future research—the impact of aggressive marketing by the food industry on the brain. As literature in this area grows, it may play a key role not only in the development of more effective intervention but also in encouraging environmentally focused policy initiatives to turn back the tide of childhood obesity. ■

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Anti-N-methyl-D-aspartate Receptor Encephalitis: What's in a Name?

In 2009, Dalmau et al[1] described the first cohort of children with anti–N-methyl-D-aspartate receptor (NMDAR) encephalitis. Their article provided a name for a previously unknown, severe pediatric encephalitis, manifested as behavioral and psychiatric symptoms in combination with dyskinesias, dystonia, seizures, progressive somnolence, and occasionally autonomic instability, notably hypoventilation and variable fever. The authors detailed the differences in presentation from adults. They also pointed out that most children with anti-NMDAR encephalitis do not commonly harbor neoplasms, unlike adults with the disease. The etiology was unclear. They reported some early success treating these children with immune-modulating therapy. Today, anti-NMDAR encephalitis is diagnosed more often than any single viral encephalitis in the US.[2]

See related article, p 850

Ultimately, however, as more children are being diagnosed with the disorder, the questions remain: What is pediatric anti-NMDAR encephalitis? Is it a disease? A syndrome? What is/are the cause(s)? Has anti-NMDAR encephalitis always existed, or is it new? Certainly, we know from the work by Dalmau et al,[3] and reproduced by others,[4] that the putative antibody binds to an extracellular epitope region of the N-terminal domain of the NR1 subunit of the NMDAR, causing a pronounced and specific decrease of NMDAR protein at synapses. We know that pharmacologic antagonists of NMDARs produce a similar profile of symptoms (eg, psychosis, agitation, memory disturbance, unresponsiveness). We have hints that the disease may be immune-mediated, given the high ratio of B cells to T cells, the presence of plasma cell infiltrates on pathologic specimens, and the dramatic response to immune

EEG	Electroencephalography
HSE	Herpes simplex encephalitis
NMDAR	N-methyl-D-aspartate receptor

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