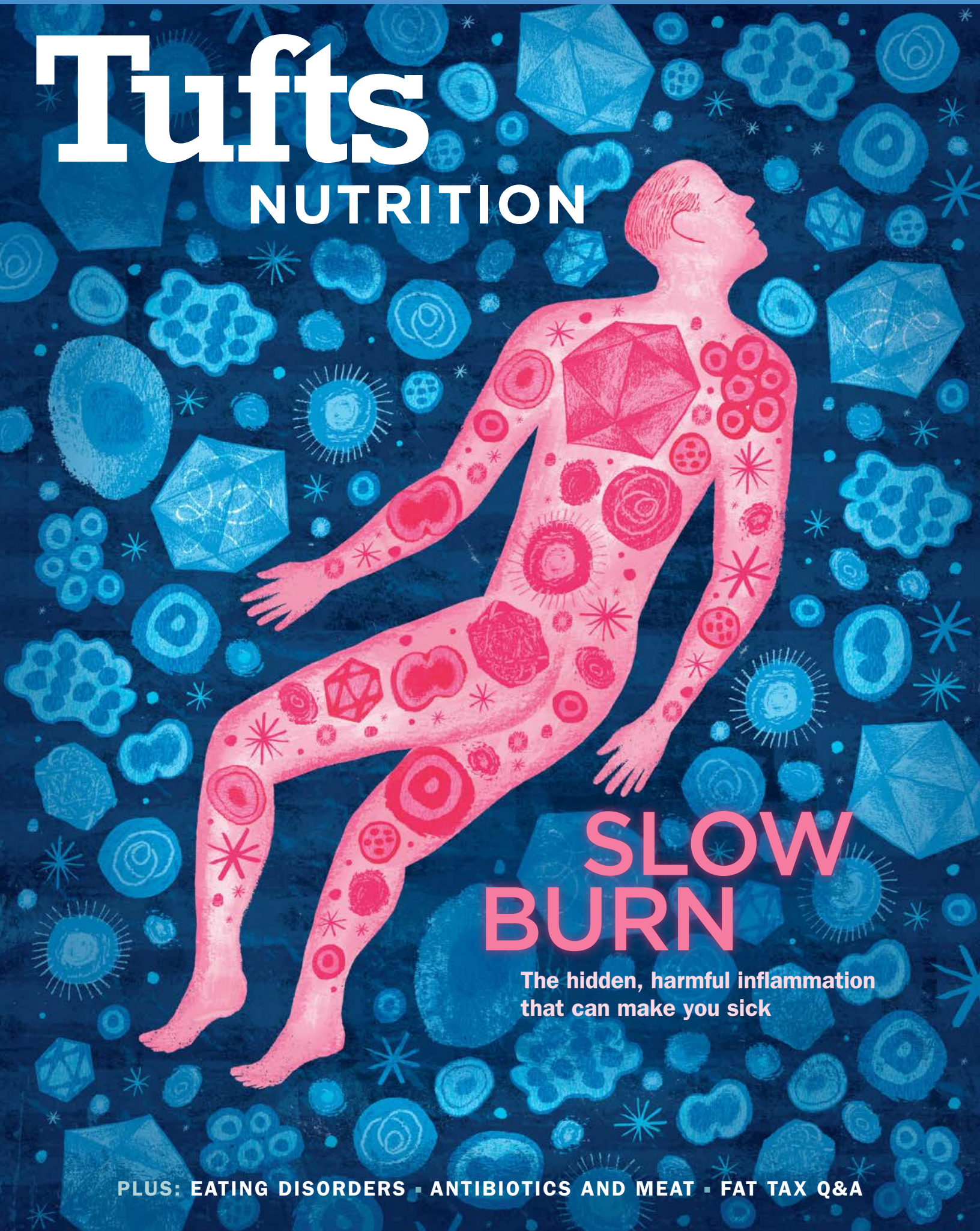
MAGAZINE OF THE GERALD J. AND DOROTHY R. FRIEDMAN SCHOOL OF NUTRITION SCIENCE AND POLICY **SUMMER 2012 VOL. 13 NO. 2**

Tufts NUTRITION



SLOW BURN

The hidden, harmful inflammation that can make you sick

PLUS: EATING DISORDERS ▪ ANTIBIOTICS AND MEAT ▪ FAT TAX Q&A

Q: Are Nutrition Bars Really All That Healthy?

For this installment of "Ask Tufts Nutrition," Alexis Madej, N08, a clinical dietitian at the Frances Stern Nutrition Center at Tufts Medical Center, serves as our expert.

A: Nutrition bars conveniently slip into purses, backpacks or briefcases and can just as easily be part of a healthy lifestyle. The key is to use nutrition bars as supplements to balance a diet of whole foods, rather than as a replacement for sensible eating.

Nutrition bars fit best as snacks, rather than meals, where you should balance your plate with vegetables, fruits, lean protein, whole grains and healthy fats. Still, snacks can fill long gaps between meals, or provide an energy boost for a daily workout.

A great combination for a satisfying snack is a mix of protein and fiber, such as low-fat yogurt swirled with fresh berries, almond butter spread on pear slices or a carefully selected nutrition bar.

It is easy to become overwhelmed by the multiple varieties of nutrition bars available, with their wide range of calories and ingredients, but a bit of investigation can steer you away from glorified candy bars. Look for a nutrition bar that meets the following criteria:

INGREDIENT LIST:

A short list of whole-food ingredients that are recognizable

More oats, whole grains, nuts and dried fruit

Less sugar and syrups

No partially hydrogenated oils

NUTRITION FACTS:

Total calories: about 120 to 250

Protein: 5 to 10 grams

Fiber: at least 3 grams

Sodium: less than 250 milligrams

Fat: less than 2 grams saturated fat, and 0 grams trans fat

Sugar: less than 10 grams

If you are always on the go, keep a bar stashed in your desk drawer, glove compartment or gym bag to ensure you always have a sensible snack on hand.

Send your questions for future installments of "Ask Tufts Nutrition" to Julie Flaherty, Tufts University Office of Publications, 80 George Street, Medford, MA 02155. Or send an email to julie.flaherty@tufts.edu.

PHOTO: KELVIN MA

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Cover illustration by James O'Brien

ANTIOXIDANT FIX

I found your article "Science, Supplements and Superfruits" (Spring 2010) very informative. I agree that the best way to get antioxidants is to eat the whole foods that are rich in these nutrients. However, what would be your next best advice for those of us who are too busy to shop for or fix produce? When I do buy produce, it spoils so easily and ends up neglected in my fridge until I throw it out. Are there less-perishable options? Some examples I have thought of are dehydrated or freeze-dried fruits and veggies that are made into energy bars. Also, I have read that cacao is a super antioxidant.

KAY KURATH
POST FALLS, IDAHO

Professor Jeffrey Blumberg, Ph.D., director of the antioxidants lab at the Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts, responds:

It is easy to achieve a high intake of antioxidants while expending little effort in shopping or food preparation: Just pick colorful, ready-to-eat fruit (such as apples, blueberries, plums and oranges) and vegetables (such as raw broccoli, carrots, celery, and red radishes) and whole grains (such as whole rye bread, whole-wheat crackers and whole oat cereals). You will have less worry about these foods being perishable if you buy them in small quantities and eat them daily.

When choosing beverages, pick black or green tea and 100 percent fruit juices. Dehydrating or freeze-drying will concentrate the antioxidants in fruits and vegetables, but read the label of energy bars carefully to determine just how much of these ingredients are actually in the product. More simply, you can pick dried fruit (figs, prunes and raisins) and nuts (their skins are rich in antioxidants). When you do cook, remember that herbs and spices (such as cinnamon, ginger, oregano and turmeric) also provide highly concentrated sources of antioxidants. Importantly, remember that there are thousands of dietary antioxidants, so don't focus on getting a few "super antioxidants," but instead enjoy as wide an array of fresh, whole-plant foods as possible.

POWERFUL WORDS

Wanted to congratulate you on your fantastic and very thorough article "Power Play" (Summer 2011). The topic is of particular interest to me, because I am a personal trainer, and the people I'm interested in helping are women in midlife. It's so important to work on building and strengthening muscle (and bone) and maintaining or improving power. Again, thanks for an informative piece.

KATHLEEN ENGEL
BLOOMFIELD, NEW JERSEY

WHOSE PLATE?

I am sure I qualify for the MyPlate for Older Adults ("Eat Well, Age Even Better," Winter 2012) as I am 83. I am wondering where this age group starts. Do you consider retirement age a starting point?

GWENN TRUAX
ENCINITAS, CALIFORNIA

Tufts Nutrition responds: Although the graphic is aimed at people age 70 and older who are living independently, the foods depicted would be healthy options for adults at any age.

TALK TO US

Tufts Nutrition **welcomes letters with concerns, suggestions and story ideas from all its readers. Address your correspondence, which may be edited for space, to Julie Flaherty, Editor,** ***Tufts Nutrition,*** **Tufts University Office of Publications, 80 George Street, Medford, MA 02155. You can also fax us at 617.627.3549 or email julie.flaherty@tufts.edu.**

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The mission of the school is to provide an opportunity for talented and passionate individuals to lead science, education and public policy to improve nutrition and health.

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We're online. Check out the magazine and the world of Tufts Nutrition at http://nutrition.tufts.edu.

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The Ties that Broaden

THIS IS MY FIRST COLUMN for *Tufts Nutrition*; however, I am no stranger to the magazine. As director of the Jean Mayer USDA Human Nutrition Research Center on Aging (HNRCA) at Tufts and a longtime laboratory director, and as a faculty member of the Friedman School of Nutrition Science and Policy, I have valued the information the publication shares with its readers for many years.

The HNRCA and the Friedman School have a mutually beneficial relationship at Tufts. For more than three decades, the school and the center have partnered to conduct research and educate students. As the largest research center in the world devoted to studying the role of nutrition in healthy and active aging, the HNRCA is one of the six human nutrition research centers in the United States supported by the USDA Agriculture Research Services. Our work contributes to research in human nutrition and aging as well as to the life sciences at Tufts University. From the inception of the school, Friedman School students have pursued practical training, carried out Ph.D. research and participated in seminars, journal clubs and other educational activities at our state-of-the-art facility on Tufts' health sciences campus in Boston.

Many of our scientists hold faculty appointments at the school and contribute to its dynamic and rigorous educational enterprise by advising students and participating on faculty committees. At the same time, many Friedman School faculty members, including Interim Dean Robin Kanarek, hold scientific appointments at the HNRCA and contribute to its research endeavors.

The cover story in this issue (see page 16), which looks at how nutrition and inflammation are closely linked to several age-related diseases, is an excellent illustration of that collaborative spirit in action. The laboratory directors whose research is highlighted in the article are also Friedman School faculty members, and Friedman School students have been an integral part of many of the research projects described in the piece.

Our partnership with the Friedman School and the university contributes to our study of nutrition and physical activity's impact on diseases from the cellular level to the population level. In the past, such collaborations have contributed to the creation of important dietary recommendations and impactful health policies. We expect to take even greater steps ahead, and look forward to sharing them with you in future issues of the magazine.

SIMIN N. MEYDANI, D.V.M., PH.D.
DIRECTOR, JEAN MAYER USDA HUMAN NUTRITION RESEARCH CENTER ON AGING

PHOTO: KELVIN MA

a la carte

RESEARCH IN BRIEF

Fat but Fit

Even if kids carry extra pounds, fitness has lasting health benefits

Being overweight doesn't necessarily mean that you can't shoot hoops and run bases like the rest of them. For overweight or obese children, it seems being or becoming fit may even lead to a healthier weight as they grow.

For a study published in the journal *Obesity*, Assistant Professor Jennifer M. Sacheck, Ph.D., N01, and colleagues examined the association between weight and fitness levels in first through seventh graders attending school in Cambridge, Mass. They collected data on 2,793 students over four years. Regardless of their weight, students were classified as "fit" if they passed five different fitness tests, such as a 20-yard shuttle run.

"Obese and overweight girls who achieved fitness were almost five times as likely, and obese and overweight boys were two and a half times as likely, to reach a healthy weight than those who stayed underfit," said first author Adela Hruby, N10, a Ph.D. candidate at the Friedman School. "It turns out that maintaining fitness is beneficial, too. We observed that obese and overweight girls and boys who started and ended the study being fit were more likely to have a healthy weight by the end of the study."

Staying fit also benefited the healthy-weight boys and girls; they were more likely to maintain their weight than those students who declined from fit to underfit over the course of the study.

The assessments coincided with a city-wide weight and fitness initiative that prompted improvements to gymnasiums, promotion of physical activities outside of school and issuing "Health and Fitness report cards" to parents.

Of the 1,069 students who were initially obese or overweight, 17 percent achieved a healthy weight within the study period. That compares with 6 percent of students who began the study at a healthy weight and became obese or overweight.

"It is encouraging to see any kind of reversal in unhealthy weight patterns, considering Centers for Disease Control statistics indicate child and adolescent obesity rates rose approximately 13 percent between 1980 and 2008," Sacheck said.

OVERHEARD

"When people understand the consequences of not taking action, they will understand. This will require bold actions from all sections of society." —Christina Economos, PH.D., N96, NEW BALANCE CHAIR IN CHILDHOOD NUTRITION AND MEMBER OF AN INSTITUTE OF MEDICINE COMMITTEE THAT CALLED FOR A CONCERTED ATTACK ON AMERICA'S OBESITY PROBLEM BY THE GOVERNMENT, THE FOOD INDUSTRY, EMPLOYERS, INSURERS AND COMMUNITIES

ILLUSTRATION: MARC ROSENTHAL

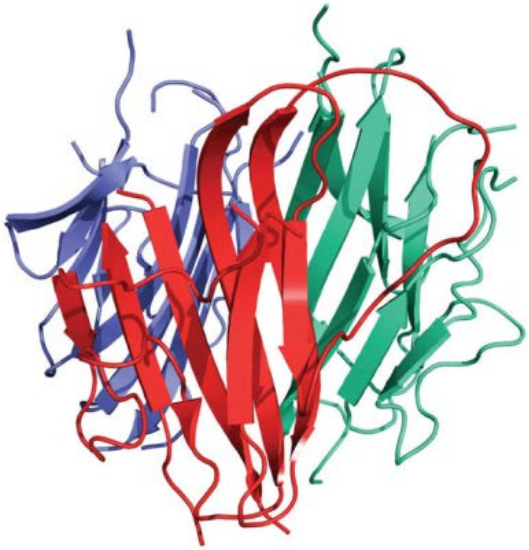
A HORMONE'S PUZZLING ROLE IN DEMENTIA

Women with high levels of adiponectin, a hormone that helps regulate the body's response to insulin, have a higher risk of developing dementia, according to a study that appeared in *Archives of Neurology*.

The aim of the study, conducted by Ernst Schaefer, M.D., director of the Lipid Metabolism Laboratory at the Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts, and colleagues, was to tease out the connection between Type 2 diabetes and cognitive decline, which have been linked in previous studies. Because people with higher levels of adiponectin have a lower risk of developing diabetes, researchers thought the hormone might also protect people against dementia. But that is not what they found.

Over 13 years, 159 of the 541 women who were tracked for the study developed some form of dementia, including 125 cases of Alzheimer's disease. The researchers found that having increased levels of adiponectin raised the women's risk of dementia by 60 percent and of Alzheimer's by 90 percent. Not enough men were tracked to determine whether the same held true for them.

The researchers point out that the adiponectin isn't necessarily causing the dementia. They write that the levels of the hormone may have risen as a "protective response to vascular damage or changes in brain morphology," conditions that some of the women may have had before the start of the study.



A molecular model of the hormone adiponectin, which may have a role in Type 2 diabetes and cognitive decline.

Grain of Proof

Rice sometimes get a bad rap from the low-carb gurus. But according to Professor Eileen Kennedy, D.Sc., and Hanqi Luo, N11, people who eat rice might be setting themselves up for good health.

Using data from the National Health and Nutrition Examination Survey (NHANES) gathered between 2007 and 2008, the researchers compared the diets of more than 8,000 adults and children who reported eating rice versus those who did not. The results, which Luo, now a research manager for Valid International, presented at the Experimental Biology conference in April, showed that those who ate rice consumed more folate, iron, potassium, vitamin A, B6, B12, thiamin, riboflavin, niacin, vitamin D, phosphorus, magnesium, copper and zinc.

Rice eaters also consumed a lower percentage of calories from fat and saturated fat compared to those who did not eat rice. In addition, adults who ate rice were less likely to be overweight or obese.

The study included people who ate brown and wild rice, which are whole grains, as well as fortified white rice.

ILLUSTRATION: LAGUNA DESIGN/PHOTO RESEARCHERS, INC.; PHOTO: ISTOCKPHOTO

The Sip that Strips

Some foods and beverages eat away at protective tooth enamel

URBAN LEGEND HAS IT THAT A CAN OF COKE WILL STRIP THE RUST OFF A nail or take the tarnish off a penny. We'll leave those experiments to *MythBusters*, but one thing is certain: Colas, sodas, sports drinks, wine and even fruit juices can damage teeth.

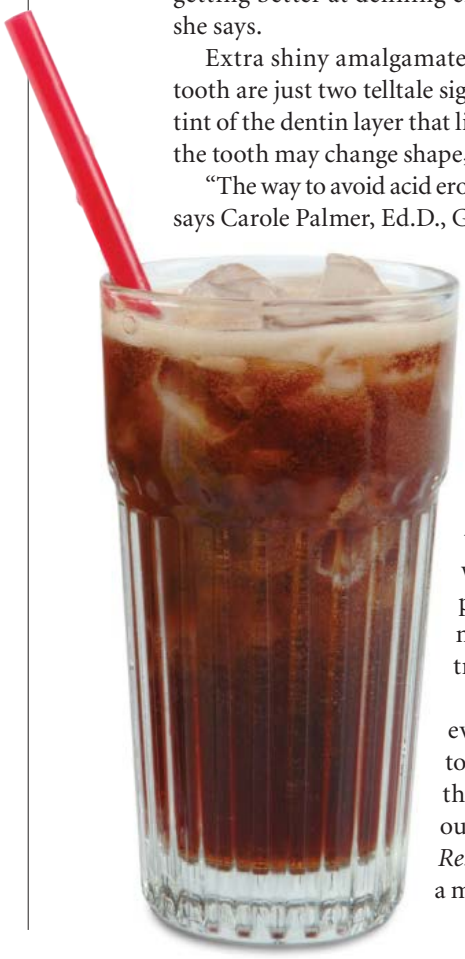
It's not just the copious amounts of sugar in these drinks that cause problems. Most sodas contain phosphoric acid, a preservative, or citric acid, for flavor. These chemicals lower the pH levels in soft drinks to about 2.5 (on a scale where 7 is neutral, and lower numbers indicate increasing acidity). Vinegar and wine score almost as low as soda, while lemon and lime juice, with a pH of less than 2, are nearly as corrosive as battery acid.

That's bad news for tooth enamel, which begins to dissolve in just slightly acidic conditions. Repeated exposure to acids will strip the teeth of their protective enamel, leaving them vulnerable to cavity-causing bacteria.

It's not clear how many people suffer from acid erosion, but it could be as high as 20 percent and is likely increasing, says Athena Papas, Ph.D., DMD, J66, professor of public health and community service and cohead of the Division of Geriatric Dentistry at Tufts School of Dental Medicine. "We are getting better at defining erosion, and it's becoming much more obvious," she says.

Extra shiny amalgamate fillings or fillings that stand up higher than the tooth are just two telltale signs of acid erosion, she says. Eventually, the yellow tint of the dentin layer that lies beneath enamel may begin to show through, or the tooth may change shape, losing its structural integrity.

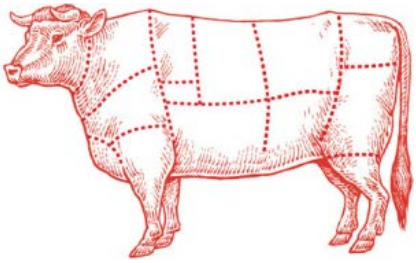
"The way to avoid acid erosion is to avoid frequent contact of teeth with acid," says Carole Palmer, Ed.D., G69, who heads the Division of Nutrition and Oral Health Promotion at the dental school and is a professor at the Friedman School.



Avoiding low pH products, such as soft drinks and sour candies—and even chewable vitamin C tablets—is definitely a step in the right direction, says Palmer. But many acidic foods, including fruits and vegetables, are part of a healthy diet. So researchers suggest, for example, using a straw to minimize contact between acidic beverages and tooth surfaces. Chasing acidic drinks or meals with plain water or milk also will help restore the pH balance in the mouth. Not eating in between meals can help, too, because it allows saliva to neutralize acids in the mouth.

And that often-dispensed advice to brush after every meal might not be so well advised. Using a toothbrush in a low pH mouth may do more harm than good, spreading enamel-eating acid throughout the mouth. A 2006 study in the journal *Caries Research* suggests that waiting 30 to 60 minutes after a meal to brush will give saliva a chance to do its job.

—JACQUELINE MITCHELL



Local Food Bottleneck

With consumers clamoring for locally pastured steak or a pork chop from a nearby organic farm, a common lament among New England farmers is that there aren't enough facilities in the region for slaughtering and processing their livestock to meet demand.

To determine whether that was the case, Assistant Professor Christian Peters, Ph.D., and Chelsea Lewis, A06, N10, surveyed New England slaughterhouse owners and managers for a paper published in the journal *Renewable Agriculture and Food Systems*. They found that on average, the facilities were actually running under capacity. It turns out that the real limitations were not a lack of facilities, but the shortage of skilled labor and the nature of the demand, which is high in the fall and low in the spring and summer, when livestock are still growing.

PHOTO/ILLUSTRATION: ISTOCKPHOTO

OBESE KIDS, EARLIER TEETH

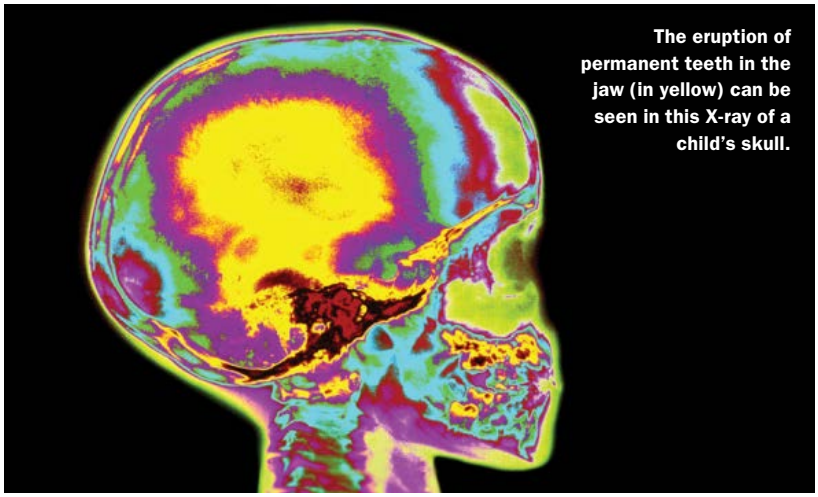
A new Tufts study finds that obese children get their permanent teeth earlier than kids who are not obese.

The early eruption of permanent teeth increases the risk for cavities, malocclusion and crowding, as well as temporomandibular joint (TMJ) disorder, says Aviva Must, Ph.D., the Morton A. Madoff Professor and chair of public health and community medicine at Tufts School of Medicine, who was the principal investigator on the study.

The research, published in February in the journal *Obesity*, "indicates the need for comprehensive and frequent oral evaluations in obese children to avoid the health pitfalls that accompany early eruption of permanent teeth," says Must, N87, N92, who is also a Friedman School professor and wrote the study with colleagues at Tufts, the Frances Stern Nutrition Center at Tufts Medical Center and HarvestPlus International Food Policy Research Institute.

The study compared the timing of tooth eruption in obese and nonobese children. The researchers analyzed data on 5,838 children, ages 5 to 14, collected as part of the National Health and Nutrition Examination Survey (NHANES) between 2001 and 2006.

They found that obese children, on average, had 1.44 more permanent teeth at any age than nonobese children. The biggest difference was seen at age 10, when the obese children had an average of three more teeth than their nonobese peers.



The eruption of permanent teeth in the jaw (in yellow) can be seen in this X-ray of a child's skull.

"All children are more susceptible to cavities when their first permanent teeth come in, because these are not fully mineralized," says Stanley Alexander, D.M.D., D75A, professor and chair of pediatric dentistry at Tufts School of Dental Medicine.

Weight is not the only factor that can affect children's tooth eruption, Alexander says, noting that sociological, lifestyle and genetic factors also play a role.

Roughly 12.5 million Americans ages 2 to 19 are obese, according to the Centers for Disease Control. These latest findings also contribute to evidence linking obesity to other aspects of accelerated growth, says Must, noting that obese children are taller before puberty than their peers, and they become sexually mature at an earlier age.

—GAIL BAMBRICK

IMPROVING THE ODDS FOR ETHIOPIA'S CHILDREN

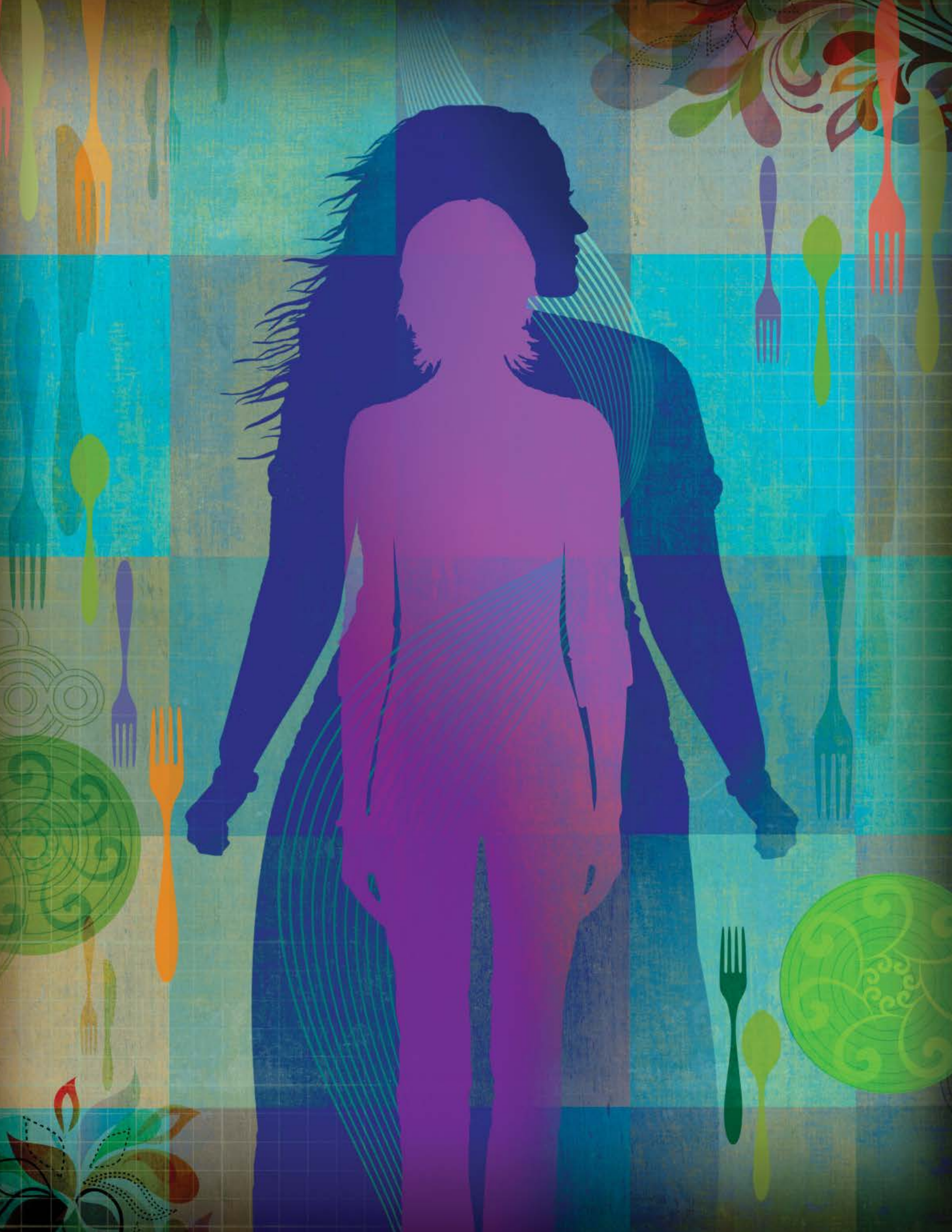
AIDED BY A $7.3 MILLION GRANT, the largest in its history, the Feinstein International Center at the Friedman School will conduct on-the-ground research aimed at breaking the centuries-old cycle of child hunger and malnutrition in Ethiopia by strengthening existing nutrition interventions.

Senior researcher Kate Sadler, Ph.D., an assistant professor, will lead a team working with families as well as Ethiopian policymakers, aid organizations and academics as part of a project directed by Save the Children and funded by the U.S. Agency for International Development. She said they will try to pinpoint why treatment programs do not reach malnourished Ethiopian children early enough, leaving them susceptible to life-threatening diseases.

"Perhaps mothers are not familiar with the warning signs of malnutrition, or they are burdened by other family responsibilities," she said. "If moderate acute malnutrition is caught in time, there are vaccinations, micronutrient-enriched foods and teachable infant feeding practices that can reverse the decline of nutrition and health in this group."

The team is expected to begin its fieldwork early next year.

PHOTO: BSIP/PHOTORESEARCHERS INC.



Obesity and eating disorders in teens spring from some of the same causes. Kendrin Sonneville, N02, makes the case for a dual public health campaign

Two Girls, One Struggle

BY JULIE FLAHERTY ILLUSTRATION BY STEPHANIE DALTON-COWAN

AFTER COMPLETING THE FRANCES STERN NUTRITION Center combined master's/dietetic internship program with the Friedman School in 2002, Kendrin Sonneville took a fellowship in adolescent nutrition at Children's Hospital Boston. There, she met with teenagers, mostly girls, who were either obese or struggling with eating disorders such as anorexia or bulimia. Polar opposites, right? The obsessive, would-be fashion models who chew ice as a meal, and the extremely overweight girls who don't put enough effort into taking care of themselves.

But that's not what Sonneville saw. "I felt there were more similarities in the work I did with those patient populations than there were differences," she says. She found that eating disorders and obesity can happen in the same person, as is the case with binge eating disorder. And although anorexia may appear the antithesis of obesity, the two often spring from some of the same causes.

Topping the risk factors for both is dieting. You would expect dieting to be exactly what we want overweight people to do. But when children diet—and nearly 56 percent of girls and 39 percent of boys ages 6 to 11 report they do—it usually backfires. In one three-year study of 15,000 adolescents conducted by Sonneville's colleagues at Children's Hospital, those who said they were dieting gained more weight than their nondieting peers.

Why the weight gain? Their metabolisms may slow down if they drastically cut calories. But the simpler explanation, one Sonneville has seen in action, is that when kids diet, it is not the kind of healthy eating dietitians espouse, such as swapping out potato chips for apple slices. Rather, they skip meals and fast—risky behaviors that lead to such overwhelming hunger that they lose all restraint and then binge eat. Or they might think a pack of Twizzlers makes a low-calorie dinner—even though these nonfilling, quickly digested foods lead to more hunger. Then there are extreme dieting behaviors, such as taking laxatives and diet pills as well as vomiting after a meal.

"Dieting is a slippery slope," says Sonneville, now director of the adolescent nutrition fellowship at Children's, where she primarily conducts research but still sees patients as a clinical nutritionist. "Virtually every case of an eating disorder starts with dieting," she says. "There is a type of dieting that is really pathological and dangerous and leads to the eating disorder track, and then there is the dieting done wrong that leads to the obesity track."

Despite their commonalities, obesity and eating disorders are usually dealt with from opposite directions: Most obesity research is focused on prevention, particularly in childhood, while eating disorders are addressed through expensive and only moderately successful medical and psychiatric treatment when—and if—they are diagnosed.

"It really seems like eating disorders don't exist in the public health arena the way that obesity does," Sonneville says. "Prevention of eating disorders is really a kind of novel concept, whereas obesity prevention is something we hear about a lot."

Sonneville, who has a Ph.D. from the Harvard School of Public Health, makes the case that our attempts to curb childhood obesity—from Body Mass Index report cards sent home by schools to the recent public health ad campaign in Atlanta that features slogans such as "being fat takes the fun out of being a kid"—should also target eating disorders. She isn't arguing that current obesity interventions are setting up kids for trouble with anorexia or binge eating. But with all the effort being put into child obesity prevention, Sonneville sees a lot that could be done simultaneously to prevent eating disorders—by keeping public health messages focused on healthy eating and feeling good, rather than making kids and parents feel guilty.

"We want to make sure all of our obesity prevention is not doing more harm than good, that we are collecting data on adverse consequences," she says. "But we should also know that we can actually help them both at the same time," she says. "If a single intervention can kill more than one bird, it's a good use of public health time and dollars."

WEIGHING THE STATS

Eighteen percent of American adolescents are considered obese, more than triple the rate reported in the mid-1970s. Compared to such epidemic proportions, eating disorders may seem like a small matter. It's true that anexoria, which is self-starvation, and bulimia, which is purging by vomiting or exercising excessively after eating, are pretty rare, affecting about one percent of adolescents. Binge eating disorder, or the frequent consumption of huge quantities of food, is more common, affecting up to 2.3 percent of adolescent girls.

Yet these are diagnosed cases, and the criteria are pretty specific. (According to upcoming guidelines by the American Psychiatric Association, for a person to be diagnosed with binge eating disorder, she would have to gorge herself, while feeling shame and a loss of control, at least once a week for three months.)

But add in cases that don't fit the clinical definitions, including people who purge after eating but still maintain a normal weight, and those who chew their food and then spit it out, and Sonneville estimates, based on her recent analysis, that 20 percent of adolescent girls exhibit disordered eating habits at some point—a percentage that rivals the prevalence of obesity.

"For something like obesity or blood pressure, it's not a dichotomous outcome," she says. "In the same way, it's not like 'eating disorder' or 'no eating disorder.' You fall somewhere on the continuum, and the goal of public health is to shift the distribution down."

Historically, eating disorder researchers and obesity experts have not worked together, in part because eating disorders are considered mental illnesses, so a lot of the research lives in psychology journals. Obesity research tends to be published in biomedical journals. Only in the last decade have researchers in both fields proposed joining forces. "It may seem that we are working at opposite ends of the spectrum, but in fact, there are lots of empirical and theoretical arguments for integrating the two, particularly integrating the prevention of the two," Sonneville says.

That means counteracting the shared risk factors. In addition to dieting, researchers have found a number of things that can set kids on the road to obesity, eating disorders or both, including exposure to media promoting the thin-is-beautiful ideal, being teased about their weight by other kids or even family members, and being dissatisfied with their bodies.

It might seem that body dissatisfaction would be a result, not a cause, of obesity. But about half of girls, including normal-weight girls, report some degree of unhappiness with their bodies. That dissatisfaction, some studies have suggested, could lead to dieting, binge eating and weight gain.

At the same time, some researchers believe that for overweight people, dissatisfaction is key to losing weight. Sonneville tells of one dietitian she worked with who would tell her overweight patients that if they wanted to eat a high-calorie food, they should take off their clothes, look in a mirror

and then decide if they *really* wanted to eat it.

"She is actually a very smart clinician, and her thinking was that this body dissatisfaction is going to motivate her patients to change," Sonneville says. "I think her intentions were good, but it's not an uncommon thought among clinicians that if you are happy with yourself, you are not likely to change your body."

So could some measure of unhappiness be a good thing? Should public health campaigns try to point out that a fat person is an unhappy person? For teens, Sonneville would argue no. For her paper soon to be published in the *International Journal of Obesity*, she tracked 1,500 overweight and obese girls (starting at about age 12) for 11 years. During that time, the girls who reported being at least somewhat satisfied with their bodies gained less weight than the girls who were dissatisfied. "All the overweight kids in the [study] are gaining weight, but those who are the most satisfied with their bodies are gaining less," Sonneville says.

Perhaps more telling is how body satisfaction affected eating disorders. Although none of the girls had an eating disorder at the start of the study, by the end, 9.5 percent of them met the criteria for binge eating disorder at least once. Yet the girls who were at least somewhat satisfied with their bodies were 60 percent less likely to develop such a disorder. In fact, among girls who were totally satisfied with their bodies, Sonneville did not see a single incidence of binge eating disorder.

"With overweight, obesity and eating disorders, I think the goal is to find out what are safe messages across the board. And that is, in part, what we did in this study," Sonneville says. Whether the message is coming from parents, teachers or a poster in gym class, "we know that if you are teaching kids to like their bodies, it is not harming them. If you have a kid who is overweight, and are sending messages about 'you are great the way you are' and 'you should love your body,' that may not be a bad thing."

"There is a type of DIETING that is really pathological and DANGEROUS and leads to the EATING DISORDER TRACK, and then there is the dieting done wrong that leads to the OBESITY TRACK."

—Kendrin Sonneville, N02

FIRST, DO NO HARM

Keeping obesity interventions from triggering eating disorders may be as simple as asking the right questions. She points to some obesity prevention efforts, such as an after-school dance program developed by Stanford University for low-income African-American girls, that track kids' weight but also monitor whether they develop eating disorders, to ensure they aren't accidently nudging them down the wrong path.

"[That] concern is real, and I think it is important that the unintended consequences of well-intentioned public health works are not overlooked," says Aviva Must, Ph.D., N87, N92, the Madoff Professor and Chair of Public Health and Community Medicine at Tufts School of Medicine. "The kind of work that [Sonneville] has done is really important in terms of keeping that front and center in policymakers' eyes."

Figuring out exactly what a public health campaign for obesity/eating disorder prevention might look like is an area wide open for research, Sonneville says. Sonneville and Must each have done research into the efficacy of BMI screenings, something a growing number of public schools are doing. In a 2008 study Must conducted in the Cambridge, Mass., public schools, she found that overall, middle schoolers were not bothered about having their weight checked at school, although, perhaps not surprisingly, overweight and obese girls felt the most uncomfortable about it. But when students reported what they would do with their BMI information, the overweight and obese girls were more likely to say that they intended to try some risky weight-control behaviors, such as fasting.

Must says the screenings seem to have value and reflect society's concern "that we have a very large public health problem and we need to face it directly." But sending home BMI report cards isn't enough. School health workers need to reach out to parents about how to talk to their child's health-care provider and what community resources are available. "The BMI reports could be the launch pad for some useful conversations," Must says. "And I think talking about body image, self-esteem and disordered eating as part of that conversation is advisable."

But if a teen's dissatisfaction with her appearance isn't a good motivator, what is? What should we say to kids to prevent them from becoming overweight?

Perhaps, Sonneville says, parents, teachers and doctors don't need to talk about weight at all. Children may be better served by creating an environment where healthy foods are the default option, physical activity is never a punishment but always done for pleasure, and adults model healthy behaviors and don't ridicule their own bodies or those of others. TN

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You Can't

Resist

Are antibiotics used in meat production fueling the growth of drug-defiant superbugs? The battle heats up

BY LINDA HALL PHOTOGRAPH BY VITO ALUIA

ANTIBIOTIC RESISTANCE COULD BRING ABOUT THE NEXT GLOBAL CRISIS—a world where "strep throat…could once again kill," warns the World Health Organization. Few experts dispute the dangers of infections that cannot be controlled. The question—debated in courtrooms and on Capitol Hill—is whether America's industrialized farms bear significant blame.

"Lots of resistant bacteria travel from farm to table," says Tufts microbiologist Stuart B. Levy, M.D. Studies have found high levels of resistant bacteria in supermarket samples of beef, pork and poultry that can cause foodborne illnesses in people who mishandle the raw meat or eat it undercooked. But meat is not the only route of transmission. Resistant strains of bacteria have been found in industrial farm workers and their families; in the air, water and soil around industrial farms; and even in the flies nearby and on cars that have traveled behind trucks bringing animals to slaughter. An estimated 90 percent of antibiotics fed to food animals are excreted in their waste, and that manure is often sold as fertilizer products, some of which are marketed as "organic compost."

Under fire is a longstanding farming practice of routinely feeding low ("subtherapeutic") doses of antibiotics to food-producing animals—not because they are sick, but to prevent disease and speed their growth. Chronic exposure to antibiotics in humans, animals and the environment, says Levy, accelerates the creation of resistant bacteria and infections against which the so-called wonder drugs of the 20th century are becoming powerless.

Levy, a modern-day Paul Revere for antibiotic resistance, has been sounding the alarm for more than three decades. He says misuse and overuse of antibiotics by doctors and patients is a major problem. Doctors prescribing antibiotics for viral illnesses such as colds, patients insisting on getting those drugs and patients failing to finish their prescriptions have all given resistant strains a better chance to take hold. But Levy says the pure volume of antibiotics used on farms—some estimates say 80 percent of the antibiotics sold in the United States are for animals, not people—has scientists concerned about agribusiness's contribution to a growing public health threat.

"Suddenly we get a report of a bacterial strain that is resistant to eight antibiotics, and you know that that occurred in animals [because it was isolated from meat samples], and it's shocking that it even occurred," says Levy, a professor at Tufts School of Medicine, president of the international Alliance for the Prudent Use of Antibiotics (based at Tufts) and author of *The Antibiotic Paradox: How the Misuse of Antibiotics Destroys Their Curative Powers*.

Levy says that one multidrug-resistant kind of staph infection—caused by methicillin-resistant *Staphylococcus aureus* (MRSA)—showed up in hospitals as early as the 1960s, before it and others like it emerged in the general population. "Now different strains are cropping up on farms. This is not a good sign," he says, because it means resistances in bacteria harbored by animals could transfer to bacteria associated with people.

UNFAIR TARGET

Agribusiness, with wide support from veterinarians, has a different perspective. George Saperstein, D.V.M., professor and chair of environmental and population health at the Cummings School of Veterinary Medicine at Tufts, says the industry is unfairly maligned, management practices are misrepresented and charges of antibiotic abuse are often erroneous and exaggerated.

Saperstein believes the industry generally uses antibiotics prudently, primarily to prevent disease from racing through flocks or herds on large-scale farms, which produce much of America's food. Management practices on such farms have come under fire for years, with charges that antibiotics are excessively and indiscriminately given to food animals so they'll gain weight and grow faster, reducing costs, and to compensate for poor nutrition and the cramped, stressful

and unsanitary conditions in which food animals spend their lives.

"The statement you hear a lot—that antibiotics are being used to substitute for good management or good sanitation—couldn't be further from the truth," Saperstein says. He is not suggesting zero problems or zero risk, but he says management practices at large farms are generally commendable.

While Saperstein acknowledges the presence of antibiotic-resistant bacteria on farms—as well as many other places—he says he has not seen enough "fingerprint" scientific evidence linking significant human disease outbreaks to multidrug-resistant bacteria strains that have emerged solely as a result of farming practices. He points to a Purdue University study that reviewed Denmark's experiences since that country phased out antibiotics as livestock growth promoters in 1999. The study suggests that changes in resistance patterns in bacteria isolated from livestock have limited influence on resistance patterns in bacteria isolated from humans.

"Lots of resistant bacteria travel from farm to table."

—Stuart B. Levy

Saperstein and others are concerned that across-the-board prohibitions of the subtherapeutic use of any antibiotic may make it more difficult for farmers to keep their animals healthy and the food they produce safe and affordable.

Consumers who worry about antibiotics in meat often confuse "residue" with "resistance." The FDA sets standards to ensure that animals brought to slaughter have undergone withdrawal periods to allow drug residues to clear their systems. Resistance poses different issues: Are there antibiotic-resistant bacteria in the meat that could sicken consumers? And are farming practices a major contributor to the pools of resistant bacteria in the environment?

All sides agree antibiotic resistance is simply a force of nature. When bacteria are exposed to an antibiotic, some of the microbes are killed, but others—ones with mutations that enable them to resist the drug's effects—survive and multiply, passing along their protective mutations. The more an antibiotic is used or misused, the more likely new, drug-resistant strains will evolve. Concerns are intensifying about the speed at which this is happening. Some strains are already resistant to multiple antibiotics. They were perhaps helped along by bacteria's recently discovered ability to exchange DNA, such that those never exposed to an antibiotic can pick up genetic material from an unrelated resistant strain and mutate into a new strain that is also resistant.

Rebecca Klein, N08, the public health and agriculture policy project director for the Center for a Livable Future at Johns Hopkins Bloomberg School of Public Health, likens aspects of antibiotic resistance to climate change: "It's relatively slow moving but potentially a huge crisis." (See "The Big, Bad Bugs," right.) Klein says consumers unaware that agriculture is implicated in antibiotic resistance are less likely to protest. "There is a certain amount of trust, thinking that if this were really a problem, the government would do something about it. I think if [the public] were more aware, they'd be demanding a shift, and that shift would take place."

On the list of things to be aware of: The Center for Science in the Public Interest documented 38 foodborne outbreaks between 1973 and 2011 in which the bacteria were resistant to one or more antibiotics. Last year 36 million pounds of ground turkey had to be recalled because of contamination by *Salmonella* Heidelberg, which is resistant to several antibiotics used in human medicine and in agriculture. That outbreak caused one death and sickened 107 people.

For the past 16 years, the National Antimicrobial Resistance Monitoring System, a collaboration of the Centers for Disease Control and Prevention, the FDA and the U.S. Department of Agriculture, has tracked data to determine whether antibiotic resistance is transmitted to humans through the food supply.

The organization's 2010 report says more than half the ground turkey samples tested carried *E. coli* bacteria that were resistant to at least three classes of antibiotics. Nearly 30 percent of chicken breast and ground turkey samples contained *Salmonella* resistant to five classes of drug, and almost 29 percent of ground beef samples carried *Salmonella* resistant to six. This means that if a consumer gets sick, there are fewer and fewer drugs to treat them. One strain found in ground turkey samples, *Salmonella* Albert, was resistant to all eight classes of antibiotics for which it was tested.

Studies have even found significant amounts of resistant bacteria, including MRSA and *E. coli,* in meat labeled as raised without antibiotics. Some speculate the meat might have picked up the bacteria from workers or at processing plants if the equipment was not sterile. Because the creation and transmission of resistant bacteria amount to an invisible web that scientists are still trying to fully

The Big, Bad Bugs

Methicillin-resistant *Staphylococcus aureus*, commonly known as MRSA, kills an estimated 19,000 people in the United States each year and sickens another 76,000. According to the World Health Organization, 440,000 new cases of multi-drug-resistant tuberculosis emerge annually, causing at least 150,000 deaths worldwide. There are reports that some strains of TB and gonorrhea, which infects 600,000 Americans each year, have rendered ineffective almost all available antibiotics.

Also of concern is the fact that almost no new antibiotics are being developed to replace the drugs that have lost their "firepower," in the words of Tufts microbiologist Stuart B. Levy, M.D., an international expert on antibiotic resistance.

Whereas 13 classes of antibiotics were created between 1935 and 1968, only two new classes have been developed in the 44 years since, as major pharmaceutical companies invest in drugs for such chronic conditions as high cholesterol, arthritis and depression. At a congressional hearing in March on legislation proposed to provide incentives for developing new antibiotics, the Infectious Diseases Society of America reported "an increasing number of patients with serious and life-threatening antimicrobial-resistant infections…against which we have frighteningly few effective therapeutics available." —L.H.

untangle, activists say it's time to curb antibiotic use on farms and in all of society.

"One of the things we say is that you can't shop your way out of this problem," says Sarah Borron, N07, a researcher at the nonprofit Food and Water Watch. "It is important to buy local, organic…and it's certainly good to support producers who are choosing not to use antibiotics," she says, but the most powerful avenue for change is in the hands of the government, which can mandate restrictions on antibiotic use.

WHERE ARE THE REGULATORS?

Public health and consumer advocacy groups focused on agriculture's role in drug resistance are increasingly frustrated by what they say is government's failure to act, so frustrated that they filed suit in an effort to force more regulation. In March a federal judge sided with those groups and ordered the FDA to follow through on restrictions against two classes of antibiotics widely used in agriculture—tetracycline and penicillin. More than 35 years ago, the FDA proposed prohibiting the use of these two drugs to promote animal growth because of concerns about antibiotic resistance. But the agency backed off, critics say, because of political and industry pressure. Now the court has ordered the FDA to proceed with that ban, although the drug manufacturers first will have the opportunity to prove their use in agriculture is not creating resistances that are harmful to public health.

In the coming months and years, this and other developments may substantially change how farmers raise the animals that produce our food. In April the FDA announced that it is asking drug makers to voluntarily change the labels that show how their antibiotics can be used on animals, eliminating their use for promoting growth while retaining their use in treating and controlling disease. And instead of allowing farmers to buy antibiotics over the counter in bulk, as they have done since the 1950s, the FDA is recommending that farmers get prescriptions for the drugs from veterinarians. The aim of these federal "guidance documents" is to compel farmers to prove they need to treat or prevent specific illness in their animals, rather than use antibiotics indiscriminately to boost production. The FDA says it has received commitments from drug makers for its voluntary plan, which would be phased in over three years.

Klein says the federal ruling is encouraging because "historically, it has seemed that the power and money from industry have won out [over] laws and regulations." She is disappointed, however, that the FDA produced guidance documents, which "are just that, *guidance* documents. If we are going to protect public health, actual regulations need to be in place with funding to ensure compliance."

Borron's assessment is more direct: "Anything less than a complete ban on the subtherapeutic uses of antibiotics in livestock is insufficient."

At the congressional level, proposed legislation would force the FDA to retract approvals for animal feed uses for any of seven classes of antibiotics determined to create resistances harmful to human health. But the legislation, the Preservation of Antibiotics for Medical Treatment Act, has been repeatedly introduced since 2003 and has yet to make it to a vote. Fingers point at intense lobbying by big agriculture and big pharma.

Also under the microscope are scattered restrictions the FDA has already imposed. In 2005 the agency banned the use of a class of antibiotics called fluoroquinolones in poultry production. The FDA said the drugs' use in poultry production had spawned foodborne fluoroquinolone-resistant Campylobacter infections in humans, a gastrointestinal illness that can be life-threatening in patients with compromised immune systems.

Just this January the FDA restricted another class of antibiotics, called cephalosporins, from being used indiscriminately in animal agriculture for disease prevention and growth promotion. "It is critical to preserve the effectiveness of these drugs," the order said. Cephalosporins are used to treat pneumonia, strep throat and other infections in humans.

Yet cephalosporins and fluoroquinolones were not as widely used in animal agriculture as are tetracycline and penicillin. "The FDA seems to be sending mixed signals," Borron says. "On one hand, it has banned subtherapeutic uses of two major classes of antibiotics [fluoroquinolones and cephalosporins], but it has taken a lawsuit to make the FDA address a proposal to ban the same uses in two other major classes [tetracyclines and penicillins]. And the FDA currently insists that voluntary efforts in cooperation with industry will work, all while new scientific evidence calls into question whether producers are even following the legal bans," Borron says.

That new evidence, a study released in April by the Center for a Livable Future and Arizona State University's Biodesign Institute, found that fluoroquinolones are still showing up in feather meal, a byproduct of poultry production used as feed for farm animals and fish. The study, in which investigators examined samples from the United States and China, found traces of pharmaceuticals and personal-care products, including caffeine and the ingredients in Tylenol, Benadryl and Prozac, which leads George Saperstein, the Cummings School veterinarian, to question whether some of the drugs were introduced not at farms but by renderers who process animal parts into fertilizer and feed.

Saperstein nevertheless advises the farm industry to make changes ahead of regulation: "If the consumer is given the impression that the industry is doing something wrong and the FDA is trying to stop them," it is more damaging than "proactively saying we're going to stop using subtherapeutic antibiotics.

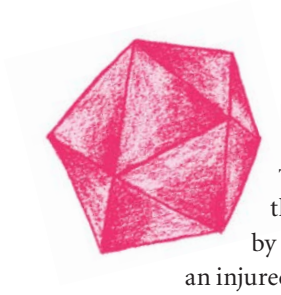
"Agriculture has always rapidly responded to consumer preferences….it's a free-market product," and farmers want to please consumers, he says. Changes in antibiotic use will create incentives for new technologies and alternatives, including more and better vaccines, he says. But Saperstein worries that some small farmers, especially those in remote locations with less access to a dwindling number of veterinarians who specialize in caring for large animals, might not "survive the shakeout" of certain mandates and financial pressures.

Klein sympathizes with the challenges farmers face. "We need laws that protect human health," she says, "*and* we need to find ways to support farmers' transition to production methods that protect essential drugs and biodiversity." TN

Linda Hall is a freelance writer in Hopkinton, Mass.

Slow Burn

Chronic inflammation underlies many of the illnesses we dread, including cancer and heart disease. Can nutrition help put out the fire?

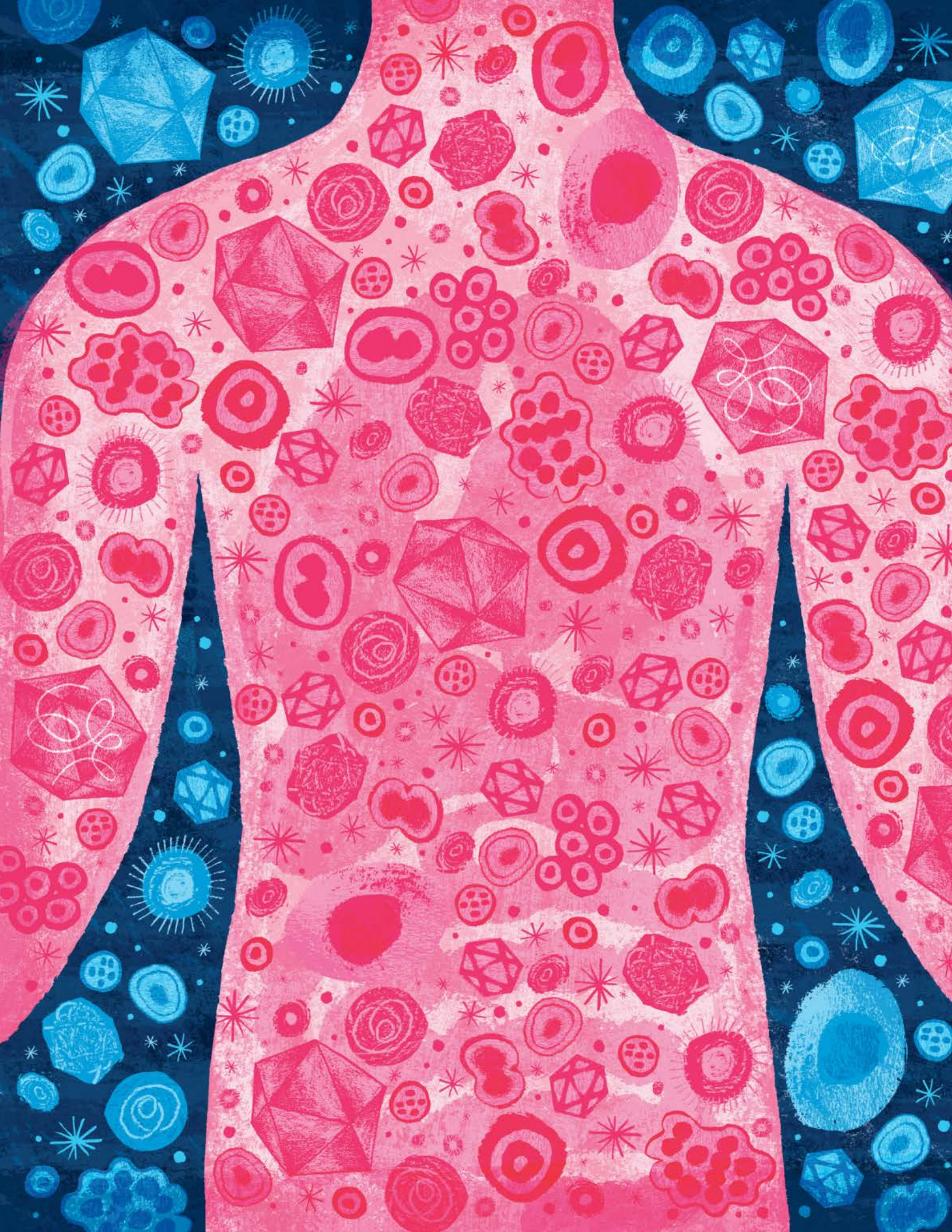


BY JULIE FLAHERTY ILLUSTRATION BY JAMES O'BRIEN

THE IMMUNE SYSTEM IS SUPPOSED TO BE OUR GREAT DEFENDER, THE ARMY that protects us against invading pathogens and rebuilds tissues damaged by injury. One of its master tactics is inflammation, where the body floods an injured or infected site with plasma and white blood cells, causing the familiar swelling, redness and don't-touch-me-there tenderness. Without it, wounds would never heal, and germs would win the war.

But sometimes the inflammation-signaling cells that are supposed to fall back at the end of their mission keep fighting, attacking an enemy only they can perceive. What keeps the battle going? Recurring irritants, such as cigarette smoke, for one. Or, as we now know, carrying too much extra weight, or simply getting older.

It doesn't mean your insides swell and throb the way your toe does when you stub it. Chronic, low-grade inflammation is not the kind you can feel, or even something you can see under a microscope. Instead, inflammatory molecules circulate at such low levels that only very sensitive tests can detect them. A full-blown infection, for example, might shoot your blood levels of C-reactive protein (CRP), a molecule released by the liver as part of the immune response, from 0 milligrams per liter up to 1,000 mg/L. Chronic inflammation, on the other hand, might register a benign-looking 3 mg/L. "It's very subtle," says Professor Joel Mason, M.D., director of the Vitamins and Carcinogenesis Laboratory at the Jean Mayer USDA Human Nutrition Research Center on Aging (HNRCA) at Tufts. "It's inflammation on a biochemical level."



But even at such a slow burn, this silent war takes a toll on the body. A CRP level of 3mg/L is enough to triple your risk of heart disease. In fact, chronic inflammation is connected to many of the illnesses we see more often as we age.

"[Inflammation] is thought to be an important basis not just for cancer, but for insulin resistance and diabetes and atherosclerotic disease and any number of other conditions," Mason says. "There is a lot of research going on into what role inflammation plays in a lot of the chronic degenerative diseases that our society falls prey to."

If inflammation is the common denominator, then finding a way to dampen it through nutrition could have far-reaching health consequences. (See "The Search for Foods that Soothe," below.) Research already suggests that inflammatory cooling properties may be what makes the fish-and-oil-rich Mediterranean diet effective in preventing cardiovascular disease and explain part of the heart-healthy power of oats. Eating fruits and vegetables, which we already know decreases the risk of cancer, may work by blocking inflammation.

Professor José Ordovas, Ph.D., who directs the Nutrition and Genomics Laboratory at the HNRCA, cochairs a cluster of researchers throughout the center who are looking at how inflammation leads to disease and how what we eat may tamp it down. Scientists have been making steady progress, he says, by studying the inflammation that underlies many diseases, rather than concentrating solely on individual illnesses.

"What happens sometimes is that we focus on the branches, because they are easy to work with, and we don't dig to reach the origin or the roots of the problem," he says.

THE OBESITY CONNECTION

It seems like the ultimate betrayal—the immune system that is supposed to defend us ends up attacking us. "The inflammatory response, in part, was put there to help us respond to various bodily insults, whether that insult is an infection or wound," Mason says. But many people have come to live in a way—overstuffed and sedentary—that they weren't meant to live. "Man wasn't obese as he was evolving over tens of millions of years," Mason argues. "And I don't think our body was designed to have this sustained inflammatory response that would persist for months and years, as it does apparently in obesity."

Everyone knows that weight gain and diabetes are closely linked. It is only more recently that researchers have seen how inflammation may be one of the bridges that connect them.

"With obesity, there is a release of fatty acids and other factors that fuel inflammation and insulin resistance," says Atkins Professor in Metabolism and Nutrition Andrew Greenberg, M.D., director of the HNRCA's Obesity and Metabolism Laboratory.

For a long time, scientists thought that the extra fat we carry on our bodies was fairly inert stuff—just more baggage to slow us down. But more recent studies, including groundbreaking research by Greenberg, have shown that fat is actually a hormonal powerhouse. As people gain weight, their fat cells grow larger. When that happens, the cells churn out several inflammation-inducing proteins, known as cytokines. And they do it at a surprising rate. One of these cytokines is Interleukin-6. You usually find IL-6 when the body's immune system is doing its day job, fighting an infection or trying to heal a burn, for example. But one study found that obese people had 10 times as much IL-6 in their fat tissue as normal-weight people. IL-6 and other inflammatory factors "block insulin's ability to signal in the cell," Greenberg explains, which is one way insulin resistance, the precursor to diabetes, can develop. This

The Search for Foods that Soothe

There is some evidence that what we eat can affect inflammation. Perhaps not coincidentally, many of these foods are the same ones you've heard recommended as defenders against heart disease and cancer.

Some studies have shown that eating a meal high in saturated fat or trans fat causes inflammation markers to shoot up, if only temporarily. But unsaturated fats, and omega-3 fatty acids in particular, seem to be protective. Studies that look at groups of people over a long term have seen connections between omega-3 intake and inflammation-causing molecules in the blood. For example, a study of 727 postmenopausal women in the Nurses' Health Study found those who had the highest consumption of omega-3s, found in fatty fish, walnuts, flaxseed and canola oil, had lower levels of CRP, that immune responder, and IL-6, that inflammation-producing protein. Why would omega-3s fight inflammation? At least one study has shown that omega-3s reduce the expression of inflammatory proteins in cells that line our blood vessels.

Choosing carbohydrates that take longer to digest and do not cause spikes in blood sugar, such as vegetables, beans and whole grains, seem to be beneficial in reducing inflammation. One study, published this year in the *Journal of Nutrition*, found that overweight people who ate a diet of such foods reduced their CRP levels, whether they lost weight or not.

Oats are one whole grain that seem to have special health properties. The consumption of oats, oatmeal and oat bran, even for a short period, has been shown in most studies to reduce total blood cholesterol and LDL cholesterol (the bad kind), the main risk factors for cardiovascular disease. Scientists mostly credit this to

"Age-associated inflammation has been identified as an important contributor to many of the age-associated diseases, including Alzheimer's, osteoporosis, loss of muscle mass and infection, in addition to cancer and cardiovascular disease."

—Simin N. Meydani

increase in inflammation appears to be a necessary step on the path to diabetes.

Enlarged fat cells bring on inflammation for a number of reasons. Engorged fat cells release fatty acids into the blood, which seem to spur an inflammatory response. People who carry a lot of weight also tend to have high numbers of macrophages in their fat. Macrophages are immune system weapons that normally gobble up pathogens or dead cells. Researchers believe the body sends the macrophages to clean up dead fat cells, the numbers of which increase dramatically with obesity. Once there, the macrophages infiltrate the fat and start sending out pro-inflammatory signals. In laboratory studies, obese mice that had excess macrophages started making a lot more insulin, and eventually developed insulin resistance.

GATEWAY TO CANCER

Inflammation might also explain, in part, the connection between being fat and getting cancer. Scientists have long known that if you carry extra weight, you are more likely to get cancer. And you don't need to be obese. "Being obese increases your risk of developing colon cancer by two- or threefold," says Mason, who has done numerous studies on colon cancer. "But just being overweight increases your risk by 20 percent."

Now scientists suspect that it is the inflammation brought on by obesity that contributes to that risk. Certain cancers have a direct connection to inflammation. Cigarette smoking, for example, irritates the lungs, which leads to inflammation and lung cancer. The human papillomavirus causes a chronic infection, and the subsequent inflammation leads to cervical cancer. And people with chronic inflammatory bowel disease have a five- to sevenfold increase in their risk for developing colon cancer.

But obesity in and of itself seems to promote cancer. Take liver cancer. The number of cases of liver cancer in the United States has doubled in the past 25 years, although researchers are unsure why. The usual causes—excessive alcohol use and viruses such as hepatitis B and C—have not increased. One thing has, though: obesity. When a person gets fatter, so does

oats' soluble fiber, which interferes with the reabsorption of bile acid in the gut and reduces cholesterol levels.

But oats also contain special compounds called avenanthramides that seem to play a role in reducing inflammation. Avenanthramides are not found found in other cereal grains—they are a part of the oat plant's own defense mechanism, produced in response to environmental stresses such as harsh weather. Professor Mohsen Meydani, D.V.M., Ph.D., director of the HNRCA's Vascular Biology Laboratory, discovered that oat extract that was high in avenanthramides could reduce the number of inflammatory signals put out by cells that line blood vessels. Add in the fact that oats also have been shown to improve the function of blood vessel cells (which help regulate blood pressure and blood flow), and it seems oats have the potential to not only decrease plaque that causes bad cholesterol in the blood, but dampen the inflammation that leads to atherosclerosis.

Meydani has also looked at a possible mechanism by which oats may reduce the risk of colon cancer. He looked at the cells that line the colon, and found that avenanthramides, in part because of their anti-inflammatory properties, inhibited the growth of cancer cells, but left healthy cells alone.

Fruits and vegetables are the cornerstone of any healthy diet. Yet more evidence for their power comes from a 2008 study by the HNRCA's Vitamin K Laboratory. Looking at data from 1,381 participants in the Framingham Offspring Study, the Tufts researchers

his liver, and that fat infiltration can cause the liver to become inflamed. In laboratory studies of rats that were given a small amount of a liver-specific carcinogen, obese rats showed more inflammation markers and more precancerous lesions in their livers than regular-weight rats, says Professor Xiang-Dong Wang, M.D., Ph.D., N92, director of the HNRCA's Nutrition and Cancer Biology Laboratory, which performed the experiments.

How does it happen? Joel Mason has discovered one route by which obesity-induced inflammation spurs cancer, at least in the colon. The lining of the colon is one of the most rapidly proliferating tissues in the body, producing tens of millions of new cells every day. "About every four or five days, you have an entirely new lining in your colon," Mason says. The destruction of old cells and the creation of new ones is orchestrated by cascades of intercellular signals. One of them, the Wnt signaling pathway, is integral to colon health. But when this pathway is overactivated, it can lead to the uncontrolled cell growth that is cancer. "More than 85 percent of all human colon cancers are thought to arise because of overactivation of this pathway," Mason says.

What triggers the overactivation? The inflammation brought on by obesity, for one. In studying the colons of obese mice, Mason saw elevated levels of pro-inflammatory cytokines, lots of Wnt signaling and an accompanying increase in cellular proliferation.

Mason says he is not trying to argue that Wnt signaling is the sole way in which obesity increases the risk for colorectal cancer, but he does believe it could be a substantial, biologically plausible pathway.

"It takes a number of different pushes from different directions to finally get the ball rolling where cells just finally decide that they are going to become cancerous," he says. "Some of us think that they get nudged a little bit by these pro-inflammatory cytokines."

Another theory about how inflammation leads to cancer has to do with oxygen free radicals, molecules that the inflammatory response signals to kill such intruders as bacteria or viruses. Unfortunately, these free radicals tend to destroy anything they come in contact with, including healthy cells. Sometimes a free radical damages a healthy cell just enough to mutate its DNA, which can trigger a cycle of abnormal cell growth.

Typically, cells have built-in mechanisms to prevent damaged DNA from being copied. But the inflammatory response, in its good-hearted attempt to promote the creation of healthy tissue after an injury, can work against those mechanisms—and even spur new blood supplies that help to fuel the growth of the abnormal cells.

It makes sense, then, that people who eat lots of fruits and vegetables have a lower risk of cancer; colorful produce is rich in carotenoids and other antioxidants, which neutralize free radicals. But sorting out which antioxidants are actually dampening inflammation has been tricky. In studies of obese rats, Wang's laboratory supplemented the rats' diet with either lycopene—an antioxidant found in tomatoes—or tomato extract. Both helped dampen inflammation, but the tomato extract did a better job.

In this case, the purified compound was not as effective as the whole food. To Wang, this means people are better off eating more tomato sauce and leaving the lycopene supplements on the shelf. "You better get this protection from your diet," he advises.

His lab is also looking at whether consuming certain nutrients can block the cancer-inducing inflammation caused by cigarette smoke. They started by looking at a Harvard analysis of dietary intake studies, which showed that smokers who ate foods rich in a carotenoid called beta-cryptoxanthan (found in pumpkin, sweet red peppers, papayas, oranges and carrots) had a lower incidence of lung cancer.

measured blood levels of vitamin K as well as dietary intake. They then looked at 14 inflammation markers. Those people who had the most vitamin K in their blood and who reported eating more vitamin K-rich foods showed lower levels of inflammation. The researchers point out that vitamin K is found in such leafy greens as spinach, lettuce and kale, as well as in cabbage, cauliflower, broccoli, Brussels sprouts, cereals, milk and soybeans—all foods that contain an assortment of micronutrients that could depress inflammation. So it is possible that vitamin K isn't itself the inflammation fighter, but some of the other micronutrients and plant chemicals it keeps company with in those foods might be.

Of course, a combination of nutritional factors could be working together to fight inflammation. A 2008 study published in the *American Journal of Clinical Nutrition* found that people in Japan who ate a diet high in vegetables, fruit, soy, fish and yogurt had lower CRP levels.

The Mediterranean diet, which features olive oil, fish, complex carbohydrates and nuts, has been reported to be associated with a lower risk for most common chronic diseases, José Ordovas says. In the case of cardiovascular disease, this protection was attributed to the monounsaturated fats in olive oil. "However, more recently, we have learned that most of the effect could be related to the minor components present in extra virgin olive oil that confer its antioxidant and anti-inflammatory capacities," Ordovas says. "Moreover, other components of the Mediterranean diet, such as the omega-3 present in fish or found in nuts, as well as some commonly used herbs and spices, act synergistically to reduce inflammation and oxidation and thus the risk of chronic diseases. Overall, these effects translate into healthier aging."

Whether these foods make up an "anti-inflammatory diet" remains to be proven. But all of these findings support many of the current recommendations for what we should be eating anyway to stay healthy.

"Diets rich in fruits and vegetables, whole grains and fish are associated with lower risk of cardiovascular disease and cancer," says Alice Lichtenstein. With a nod to Shakespeare, she adds: "The diet you describe, by any other name, would be the same." —J.F.

"I don't think our body was designed to have this sustained inflammatory response that would persist for months and years, as it does apparently in obesity." —Joel Mason

Wang's lab took two groups of ferrets—one given beta-cryptoxanthan and one not—and exposed them to cigarette smoke. The animals that were given the supplement fared much better. "It almost totally blocked smoking-induced inflammation and precancerous lesions," Wang says. A follow-up study went a step further: It found that beta-cryptoxanthan actually prevented the growth of lung tumors in animal lung cancer models. The research was presented at the American Association of Cancer Research annual meeting in April.

HEART ATTACK TRIGGER

Chronic inflammation gained its current notoriety when it was implicated in the number-one cause of death around the world: cardiovascular disease. The old, simplistic belief was that having a high concentration of cholesterol in the blood would cause it to build up in blood vessel walls until it decreased or totally blocked the flow of blood—a clog in the plumbing, so to say. Now researchers theorize that there is more to the relationship between blood cholesterol and arterial cholesterol, and that inflammation plays an important role in promoting these blockages, or plaques, as well as in triggering chest pain, stroke and heart attack.

The process works something like this: High concentrations of LDL, the "bad" cholesterol, lodge in the lining of blood vessels. Macrophages, those scavenger cells sent out by the immune system, recognize that the LDL isn't supposed to be there, and ingest it. These cells, now puffed with cholesterol, embed themselves in the blood vessels and form the fatty streaks that are the first step in plaque formation. Inflammatory signals sent out by the macrophages encourage additional cholesterol buildup. Eventually, a fibrous collagen cap develops and seals off the plaque. These capped plaques can sit benignly for years. Only when the plaques rupture does trouble begin, and again inflammation seems to be the culprit. In laboratory tests, researchers have seen that macrophages can secrete enzymes that degrade the cap.

"Inflammation is thought to make atherosclerotic lesions less stable," explains Gershoff Professor Alice H. Lichtenstein, D.Sc., director of the HNRCA Cardiovascular Nutrition Laboratory. "If they rupture, the surface becomes thrombogenic, making it more likely a blot clot will form and clog up a vessel." If the clot blocks the flow of blood to the heart, you have a heart attack. To the brain, you have a stroke.

Simply getting older can also increase chronic inflammation.

"Age-associated inflammation has been identified as an important contributor to many of the age-associated diseases, including Alzheimer's, osteoporosis, loss of muscle mass and infection, in addition to cancer and cardiovascular disease," says Simin Nikbin Meydani, D.V.M., Ph.D., director of the HNRCA and its Nutritional Immunology Laboratory, who has worked on several studies looking at age-related inflammation over the past 25 years.

She has found that one of the important players in the aging game is a messenger chemical, prostaglandin E2, which normally helps activate the inflammatory response. As we age, our bodies tend to make more prostaglandin E2, and that excess has been associated with nerve pain, plaque rupture in the arteries, cancer and suppression of function in T cells, which are crucial to the immune system.

COX-2 inhibitors, a group of inflammatory drugs, can suppress prostaglandin production, but they can cause dangerous side effects such as stomach bleeding. So Meydani and her HNRCA colleague, Assistant Professor Dayong Wu, M.D., Ph.D., have been looking at foods or supplements that might do the same job as the drugs. For example, people who eat diets high in omega-3-rich fish produce less prostaglandin. Vitamin E was shown both in mice and humans to reduce prostaglandin E2 production and improve immune response. Studies also showed that restricting calorie intake lowers prostaglandin production while improving immune response.

So what can you do to keep inflammation at bay? The best defense may be keeping body fat in check. Weight loss has been shown to decrease people's levels of C-reactive protein, one of the markers of inflammation. A 2002 study in the journal *Circulation* tracked 25 obese, postmenopausal women who went on a weight-loss diet. They lost an average of 30 pounds, and reduced their CRP more than 30 percent.

Then again, most Americans haven't exactly excelled at keeping their bodies at a healthy weight. That's why Greenberg, Mason and others have been looking at other ways to interrupt the obesity-inflammation-disease cycle.

"We have identified various factors, which, if we block their action, blocks inflammation and promotes improved insulin and glucose blood levels in obese animals," Greenberg says. The next step will be identifying drugs—or nutrients—that can curb the inflammation and reduce the rate of obesity-associated diabetes, which is reaching epidemic proportions. TN

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PRESIDENT'S SYMPOSIUM

New Thinking About Children

BY MICHAEL BLANDING Presidential Inauguration week, in October 2011, was marked by brilliant displays of Tufts talent, oratory and regalia. But given the new president's stature as a scientist, it seemed fitting that there should also be a quieter, more scholarly event: a meeting of minds on the subject of child development. Anthony Monaco, Tufts' 13th president, wanted to prove the value of having different disciplines train their sights on the same topic—an approach that is likely to become more and more common in these parts. Accordingly, Peggy Newell, provost and senior vice president *ad interim*, organized the first-ever inaugural symposium, Advances in Child Development. The speakers included Monaco, himself a specialist in the genetics of developmental disorders, and one of his U.K. mentors, Sir Michael Rutter. They were joined by half a dozen top scientists from different schools of Tufts. Topics ran the gamut from dyslexia to tooth decay. Herewith, a digest of the talks, the full titles of which are given after each speaker's name.

WHEN GENES GO AWRY

ANTHONY P. MONACO, PRESIDENT, TUFTS UNIVERSITY; PROFESSOR OF BIOLOGY, SCHOOL OF ARTS AND SCIENCES; PROFESSOR OF NEUROSCIENCE, SCHOOL OF MEDICINE ("GENETICS OF NEURODEVELOPMENTAL DISORDERS")

It's long been known that developmental disorders run in families, but pinpointing genetic culprits is no small task. Monaco and his laboratory have taken several quite different approaches. To track down the genes associated with autism, for example, they homed in on chromosome abnormalities that occur when cells divide, causing breaks in genes or changes in their number, for example by deleting a copy. Scrutinizing those chromosome abnormalities turned up single genes, or sometimes a group of genes, that influence susceptibility to autism in 15 to 20 percent of cases.

In another case, involving a family with a rare speech and language disorder, Monaco and his colleagues narrowed the cause down to a single gene, FOXP2. An abnormality in that gene could signal whether a member of the family would inherit the language problem, even before any symptoms appeared. By studying the FOXP2 gene in songbirds, mice and monkeys, the researchers gained insights into the gene's role in vocalizations throughout evolution and how language developed in humans. "It's allowed us at least one entry point to start to dissect the biological pathways inside the brain involved in speech and language," Monaco said.

Dyslexia is another neurodevelopmental disorder with a genetic component. Monaco's team managed to identify one of four genes apparently associated with the disorder. Studies show that the gene affects how neurons form in the fetal brain. It "might be involved in the correct adherence and migration of neurons," Monaco said.

With all of these disorders, early detection can be key to improving outcomes. The contributions Monaco and other genetics researchers are making to the field eventually could lead to screening techniques that would improve diagnosis and give doctors better insight into potential treatments.

ILLUSTRATIONS: STUART BRADFORD

NATURE AND NURTURE

SIR MICHAEL RUTTER, PROFESSOR OF DEVELOPMENTAL PSYCHOPATHOLOGY, LONDON INSTITUTE OF PSYCHIATRY ("KEY PARADIGM SHIFTS IN DEVELOPMENTAL SCIENCES")

It's an old debate: heredity versus environment. Which of the two exerts the greater influence on the individual has proved to be even more complicated than once believed, according to Rutter. While psychiatrists have fixated on either biological or environmental causes of mental disorders, most such conditions now appear to stem from a complex interplay between the two.

Many of the newer findings have come from "natural experiments," studies that compare naturally occurring groups so as to isolate genetic effects—the children of twins, for example, or children born of artificial insemination versus children of donated eggs. Previously, some clinical studies suggested that ADHD or antisocial behavior may be caused by a mother's smoking during pregnancy—an environmental factor, in other words. But these newer studies have shown that children with certain genetic profiles suffer no such ill effects from their mothers' smoking, although there is an effect on birthweight. In those cases, genes trump environment.

Sometimes, the opposite seems to be true. Childhood depression was long thought to be genetically determined, but new studies show that environmental factors are twice as important as genetic predisposition in determining whether a child is diagnosed with the disease. In some cases, genes and environment can have a multiplier effect, exponentially increasing the chances of developing certain disorders. In still other cases, environmental forces can be canceled out by genetics. A variant of one particular gene, for instance, affects the vulnerability of children experiencing maltreatment, helping them develop resiliency.

All of these findings have dramatic implications for treatment—allowing better targeting of certain disorders. But first, Rutter said, doctors must abandon their old notions of nature *or* nurture in favor of a more individualized approach that sees the two as inseparable.

REWIRING DYSLEXIA

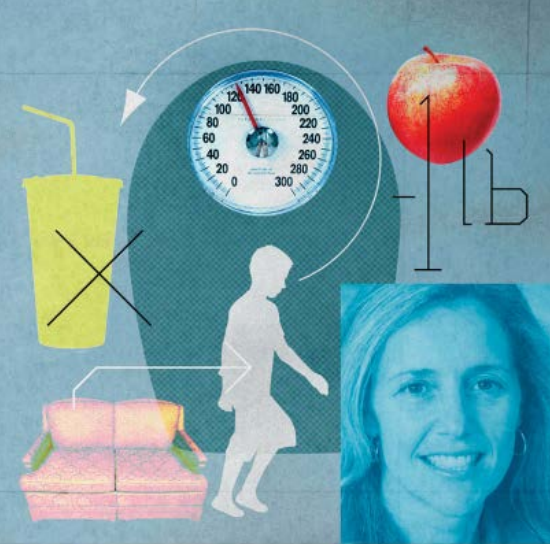
MARYANNE WOLF, THE JOHN DIBIAGGIO PROFESSOR OF CITIZENSHIP AND PUBLIC SERVICE, DIRECTOR OF THE CENTER FOR READING AND LANGUAGE RESEARCH AND A PROFESSOR IN THE ELIOT-PEARSON DEPARTMENT OF CHILD DEVELOPMENT, TUFTS UNIVERSITY ("THE EVOLVING READING BRAIN IN A DIGITAL CULTURE")

To most of us, reading seems such a basic brain function that we hardly stop to think about how we learned to do it. In fact, said Wolf, human beings aren't born with any inherent ability to read text. Rather, we all jury-rig unrelated parts of our brain—those in charge of visual recognition of shapes, phonological recognition of sounds and higher cognitive functions for comprehension and syntax—into a complex circuitry that decodes the sentence you are reading now in a matter of milliseconds. "Each new reader," Wolf said, "must create an entire reading circuit"—usually between ages 5 and 7. "It took us 2,000 years as a species to get to an alphabet," she said, referring to the evolution from symbolic writing, such as hieroglyphics or cuneiform, to the first real phonetic writing system, developed by the Greeks around 800 B.C. But a child must go through a similar cognitive evolution in just 2,000 days.

It's no wonder that this complicated process sometimes goes haywire, causing reading disorders. Brain scans have shown that many dyslexic children fail to activate areas in the left hemisphere for phonological skills that help in sounding out letters; instead, they use areas in the right hemisphere keyed to visualization. Wolf and her colleagues have devised ways to rewire those faulty pathways. Putting children through a special curriculum that breaks down each stage of the reading process into specific techniques, mimicking the sequence of activities in the brain, the researchers have seen marked improvement in both word recognition and reading comprehension.

Her latest studies look at how the

daily bombardment of technology is affecting children's reading development. "Is that going to be changing the degree to which they possess the deep reading skills?" she asked. In other words, how will all of the circuitry we've created for computers affect the circuitry we've created in our brains?



GROWING UP, NOT OUT

CHRISTINA D. ECONOMOS, N96, VICE CHAIR AND DIRECTOR, CHILDOBESITY180; NEW BALANCE CHAIR IN CHILDHOOD NUTRITION, FRIEDMAN SCHOOL OF NUTRITION SCIENCE AND POLICY; ASSOCIATE PROFESSOR OF FAMILY MEDICINE AND COMMUNITY HEALTH, SCHOOL OF MEDICINE ("TACKLING A CRISIS: A SYSTEMS APPROACH TO OBESITY PREVENTION")

Everybody knows that childhood obesity is a problem. Economos has actually been able to do something about it. A decade ago, she helped put together a community research study in Tufts' home base of Somerville, Mass. Since then, Shape Up Somerville has become a national model. Aimed at getting children to eat healthier foods and increase physical activity, the program used a "systems" approach, tackling all sides of the obesity issue at once. Parents plotted walking routes to schools, the schools switched to healthier menus and doctors were trained in more effective screening for obesity. Even local restaurants got in on the act, offering smaller portions.

After the first year, the study showed a drop in obesity that translated into about a pound per child per year compared with two neighboring towns—a significant amount over a childhood. "It's exactly what we hoped for," said Economos. When Michelle Obama launched a national campaign to combat childhood obesity, she singled out Shape Up Somerville for praise. Economos is now developing a national "playbook" for tackling the problem, recognizing that just as there is no one cause for the obesity epidemic, so there is no one-size-fits-all solution.

TREAT THE FETUS

DIANA BIANCHI, EXECUTIVE DIRECTOR, MOTHER INFANT RESEARCH INSTITUTE, TUFTS MEDICAL CENTER; NATALIE V. ZUCKER PROFESSOR OF PEDIATRICS, OBSTETRICS AND GYNECOLOGY, SCHOOL OF MEDICINE; VICE CHAIR FOR RESEARCH AND ACADEMIC AFFAIRS, DEPARTMENT OF PEDIATRICS, FLOATING HOSPITAL FOR CHILDREN ("TREATMENT OF DEVELOPMENTAL DISORDERS USING A PRENATAL GENE EXPRESSION APPROACH")

The joy of a new pregnancy is often overshadowed by The Test—routine prenatal screening for Down syndrome. If the result is positive, the parents must choose whether to continue or terminate the pregnancy. Bianchi is working on a third option: treating the condition in the womb. Her lab has been looking for drugs that, given to the pregnant woman, might counteract some of the biochemical abnormalities seen in Down fetuses. Examining RNA that floats in the amniotic fluid, Bianchi and her team found that genes in such fetuses are affected by something called oxidative stress—a byproduct of oxygen metabolism that creates destructive chemicals. The researchers plugged the gene expression profiles of the affected fetuses into a database of all FDA-approved drugs, searching for any that might stop the oxidative effects.

Sure enough, several drugs popped up. In early results, one of the drugs greatly reduced oxidative stress in cells from Down fetuses in the test tube. Now Bianchi's team is experimenting with mice. "To the best of our knowledge, this is the first time anyone has tried to use a gene expression rationale for treating a genetic condition in utero," Bianchi said. If it works, the same techniques could be used to treat other birth defects, perhaps opening up a whole new field: prenatal pediatrics.

VITAMINS FOR TEETH

HUW F. THOMAS, DEAN AND PROFESSOR OF PEDIATRIC DENTISTRY, TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE ("EARLY CHILDHOOD CARIES: THE ROLE OF NUTRITION")

The most common disease of childhood isn't asthma or diabetes. It's early childhood caries, more commonly known as tooth decay. By age 5, some 25 percent of affluent children show some signs of the disease—caused by bacteria that turn sugar into acid, which breaks down the enamel on the teeth. Among poor children, however, the disease is epidemic, affecting 60 percent of them by age 5. Some children exhibit signs of decay before their first birthday. "This disease shows no age limitations," said Thomas.

While many studies have examined the role of sugar or improper bottle feeding in causing cavities, Thomas has focused on a less intuitive cause, deficiency of vitamin D, a problem that particularly affects poor communities due to inadequate nutrition. Experimenting with mice that can't metabolize vitamin D, Thomas has shown dramatic differences in their tooth enamel compared

LEARNING FROM ANIMALS

LISA FREEMAN, J86, V91, N96, PROFESSOR OF CLINICAL SCIENCES, CUMMINGS SCHOOL OF VETERINARY MEDICINE, TUFTS ("CHILD DEVELOPMENT: LINKING HUMAN AND ANIMAL HEALTH")

"You may be wondering why you have a veterinarian talking to you at a symposium on child development," said Freeman. "It all boils down to the idea of One Health." It's the proposition that studying animals can yield insights into the treatment of human disorders, including developmental ones. The concept has been championed by Tufts and by Freeman in her research on animal nutrition.

One disease that affects both animals and humans is hypertrophic cardiomyopathy (HCM)—a heart condition that can cause sudden cardiac death in young athletes. Maine coon cats have a genetic mutation that renders them more susceptible to the disease, and Freeman has studied them intensively. An intriguing finding: some cats with the mutation get very sick and others not at all. Why? Possibly because of differences in nutrition, Freeman said.

to ordinary mice. Surprisingly, the teeth of mutant mice form their enamel coating faster than normal. The trouble is, the enamel is weak. In ordinary mouse enamel, there's a strong crystalline structure—"a beautiful prism arrangement," as Thomas called it (he displayed an electron microscope image of what looked like a wall made of neat rows of bricks). By contrast, the teeth of the mutant mice exhibited a "significant disruption of that crystal." In preliminary tests, acid dissolved the D-starved enamel much faster than the normal enamel.

While the research doesn't discount the importance of limiting sugar and good old brushing and flossing to control cavities, it does point to another dimension of tooth care that dentists should start to address.

In all sorts of animals, malnourishment during early development can interfere with such important processes as DNA repair and cell growth cycles. "Low birth weight in children can increase the risk for coronary heart disease and hypertension in later life," Freeman said. The research on HCM could open up new treatment methods both for cats and for humans.

Sometimes pets play a therapeutic role for humans. People who want to shed pounds, for example, are more successful if they are also trying to help their tubby dog or cat lose weight. Pets can even help children learn to read. "Dogs are nonevaluative, nonjudgmental listeners," Freeman said. "So children who lack confidence or lack reading skills are very comfortable reading to dogs." Freeman and her colleagues found that second-graders who read to dogs became better and more dedicated readers than those who read to other people. There is no word yet on how this affected the literacy of the dogs.

BUILDING SCHOLARS

CHRIS ROGERS, PROFESSOR OF MECHANICAL ENGINEERING AND DIRECTOR, CENTER FOR ENGINEERING EDUCATION AND OUTREACH, SCHOOL OF ENGINEERING, TUFTS ("KINDERGARTEN ENGINEERING: MOTIVATING CREATIVITY AND INNOVATION IN THE CLASSROOM")

A local first-grade teacher gave her pupils Lego blocks, gears and a mechanical motor, and asked them to make a snowplow to push Styrofoam packing peanuts out of the way. Most of the kids aced the assignment. But then she made the peanuts heavier by soaking them in water. Now only a few kids, those who had used geared wheels for more power, succeeded. "All of a sudden," Rogers recounted, "they were really interested in what gears were."

That hands-on lesson is typical of the way Rogers and his outreach team teach mechanical and mathematical principles to young children. They set a goal, encourage as many routes to that goal as possible and then let the kids keep trying until they succeed.

When kids learn with their hands, they retain more. Children who learned fractions from a blackboard did just as well on a math test as those who learned fractions by programming a Lego robot car to travel different distances. But tested again six weeks later, the Lego kids remembered almost twice as much as the blackboard kids.

Such methods aren't easy to integrate into a classroom geared to standardized tests, so Rogers and the center have developed teaching tools, training programs and online communities to help teachers adopt the techniques. Ultimately, he'd be happy if standardized tests favored creative problem-solving. "If we can try and get as much variation in the solutions as possible, instead of one right answer," he said, "then I think we've succeeded."

NOT REVOLUTI

Why the call to eat local and organic isn't speaking to everyone

BY HELENE RAGOVIN PHOTOGRAPH BY YOON S. BYUN

FOR MOST OF THE PAST DECADE, AMERICANS DISENCHANTED with the nation's food system have been advised to "vote with their forks." By filling their shopping carts and tables with organic, locally grown foods—so the conventional wisdom of this food movement goes—conscientious consumers can avoid the ethical and nutritional pitfalls of a flawed industrial system and force it to improve.

But for every shopper eagerly filling a canvas bag with heirloom tomatoes, there are dozens who have neither the money nor the ability to participate. The food movement narrative doesn't speak to the reality of inner-city residents stuck in a "food desert" miles from the nearest supermarket, or farmhands who don't earn enough to buy the very produce they spend back-breaking hours picking every day. And in that sense, the movement is leaving millions of potential allies behind.

"In order to be voting with your fork, you need to be able to buy the stuff that the food movement would have you buy," says Julian Agyeman, Ph.D., professor and chair of urban and environmental policy and planning in Tufts' School of Arts and Sciences. And while acknowledging that his middle-class status affords him the chance to follow the food movement's recommendations—"I shop at Whole Foods," he says without apology—Agyeman has also become a thoughtful critic of the food movement through his scholarship in a field that has come to be known as "food justice."

Food justice is more than making sure that food is good for your health or produced in a way that's good for the planet. It's also about ensuring fair treatment for the workers who produce the food and that high-quality, culturally appropriate food is available, regardless of a person's income, race or ethnicity. The scope of the food justice philosophy is covered in a wide-ranging collection of essays edited by Agyeman and Alison Hope Alkon, a sociologist at the University of the Pacific, called *Cultivating Food Justice: Race, Class and Sustainability* (MIT Press, 2011).

"One of the reasons I got really interested in the topic of food justice is that a lot of my scholarship is on social justice and sustainability," says Agyeman, who began his career with training in geography and botany. He has also published on the subject of environmental justice. "To me, it seems food is a great place to start our discussion."

In *Cultivating Food Justice*, "we try to focus on the difference between nutritional approaches to food and cultural approaches to food," Agyeman says. "Food justice is not just about provisioning of nutritionally appropriate food, but about maintaining the cultural significance of food." For instance, Central American immigrants who used traditional Mayan farming techniques in a South Central Los Angeles community garden were not just growing corns, beans and squash, but maintaining their culinary heritage. (Tellingly, the farm, which is pictured in the book, was later abolished when the land was sold for big-box retail development.)

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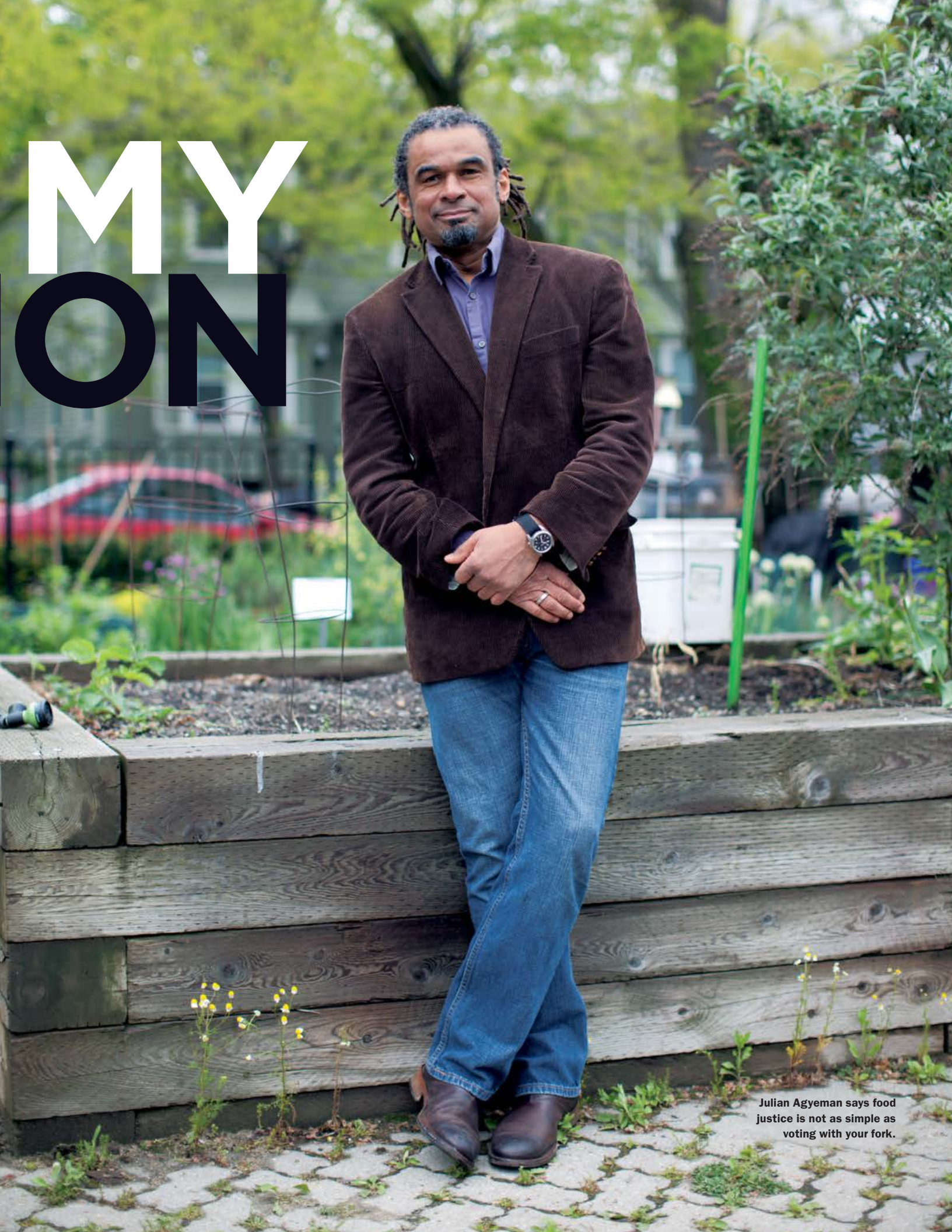

Julian Agyeman says food justice is not as simple as voting with your fork.

The concept of food justice is similar to the idea of environmental justice—"the well-substantiated claim that low-income people and people of color bear a disproportionate share of the burden of environmental degradation," Agyeman and Alkon write. Inner cities, for example, are often blighted by air pollution from highways, a phenomenon not found in suburban neighborhoods.

As a movement, food justice "has begun to take root between the cracks in busted sidewalks in some of the poorest neighborhoods in the United States," they write. One of the first, and now among the largest, of the food-justice organizations, Milwaukee's Growing Power, consists of a working urban farm that employs upwards of 65 people and educates city residents about sustainable community food systems. The nonprofit known as Just Food, begun in 1995 in New York, has helped start more than 100 community-supported agriculture (CSA) setups; trains people to run community gardens and farmers' markets; and helps supply food pantries with fresh produce, among other things.

This may not sound very different from the priorities of the food movement that Agyeman and other authors in *Cultivating Food Justice* criticize. Fresh kale? Just-picked beets? What's not to like?

Where the food movement falls short, they argue, is in several of its basic assumptions, which are largely based on race and class. Specifically, the movement has very much limited itself to mostly white, middle-class people talking to other mostly white, middle-class people ("a monoculture of PLUs—people like us," Agyeman told a group of Tufts undergraduates recently). An example: the romanticizing of an agrarian past and the concept of agriculture as, essentially, recreation. Helping dig potatoes at your CSA might bring back pleasant memories of days with grandpa on the family farm—but not so much if your grandpa was a sharecropper or a migrant worker.

One essay in the book recounts the experiences of a college student working with urban teens who were sent on a field trip to pick fruit. "The director of the youth program had said it would be a good idea for the youth to 'get their hands dirty'… [but the African-American youth] … resented the expectation to work not only for free, but also for white farmers," writes contributor Julie Guthman, an associate professor at UC-Santa Cruz, in a piece titled "'If They Only Knew': The Unbearable Whiteness of Alternative Food."

WHERE THE FOOD MOVEMENT FALLS SHORT IS IN SEVERAL OF ITS BASIC ASSUMPTIONS, WHICH ARE LARGELY BASED ON RACE AND CLASS.

Likewise, Agyeman questions the implications of the "eat local" mantra, another pillar of the food movement.

"Yes, sadly, we are disconnecting from nature, and we are disconnecting from the seasons as well," Agyeman laments. "When I was a kid in the early '60s in Britain, we didn't get food out of season. If you were eating a strawberry, it meant it was summer.

"But," he continues, "I have issues with the dogma of the local food movement that says 'eat only what *should* be grown locally.' That's a real problem in that it precludes immigrants from getting culturally appropriate foods. I can understand why people say that, but one thing the local food movement has got to come to terms with is that it cannot be prescriptive. We can't say we should only support, or not support, certain kinds of foods." In other words, the Jamaican family who wants the fresh leafy green known as callaloo and the Brazilian cook seeking manioc root in Boston shouldn't be told to re-engineer their recipes to accommodate spinach or pumpkin.

And while "eat local" seems to be unassailable from an environmental perspective, Agyeman reminds us that "food miles"—the distance food must travel from its point of origin to the point where it's eaten—shouldn't be the only measure of "greenness." When Tesco, the giant British supermarket chain, decided to source its products from as close by as possible, it seemed obvious to choose tomatoes grown in Holland, rather than those from Spain. But Dutch tomatoes are grown in greenhouses, requiring considerable amounts of energy; Spanish tomatoes, by and large, grow in the sun.

And what about the economic and social disruption to the Kenyan farmers who had been supplying Tesco with produce, specifically because the British company cultivated them as suppliers? "There are a lot of social-justice implications here as well," he says.

Nowhere does Agyeman poke at a sacred cow more than in his criticism of Michael Pollan, author of *The Omnivore's Dilemma* and *Food Rules*, and arguably the writer most responsible for popularizing the food movement. Agyeman raised a ripple of nervous laughter from the Tufts undergraduates when he took Pollan to task during his recent presentation. "We all love Michael," he says. "But Pollan really doesn't get it."

In *Food Rules*, for example, Pollan tells readers, "Don't eat anything your great-grandmother wouldn't recognize as food."

Agyeman says he understands Pollan's good intentions to steer people away from processed foods, but the advice "belies the fact that for some people in this country, their great-grandmothers were slaves and were given scraps off tables. Some great-grandmothers' food was demonized and marginalized. It just amazes me that a man as intelligent as Pollan came out with such a statement, in the sense that he's alienating a lot of people by saying that."

In overlooking the millions of people whose histories and present-day realities are absent in its rhetoric, Agyeman says, the food movement is incomplete. Using the concept of food justice, he argues, could mobilize far more people and achieve more significant results.

"I'm not trying to embarrass the largely elite food movement," he says. "The aim of the book is to look through many different lenses at food as a cultural issue and see ways that a bigger movement, a more powerful movement, can be realized if we take the cues." TN

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Sean Cash, awash in sweet treats

Weighing a Fat Tax

An economist questions whether charging more for sinful foods will get us to change how we eat by Marjorie Howard

As health-care providers and lawmakers struggle to deal with the nation's ongoing obesity epidemic, the debate continues on whether taxing unhealthy foods would encourage us to eat better. If we have to pay more for a Coke or a Twinkie, we'll back off from buying some of the soda and junk food, or so the thinking goes.

Sean Cash, Ph.D., an expert on the economics of food and the environment who recently joined the Friedman School as an associate professor, has done research to learn whether so-called "fat taxes" actually get people to change the way they eat. He's found that such taxes can result in people simply substituting other unhealthy foods to avoid the taxes. He and other researchers are also looking at what he calls "thin subsidies," ways to make healthier foods, such as fruits and vegetables, cost less.

PHOTO: JOHN SOARES

He explained his findings to *Tufts Nutrition.*

What is a fat tax?
A fat tax is food pricing geared toward influencing consumer behavior by targeting less desirable foods to make them more expensive. Usually these taxes are proposed for soft drinks or specific snack foods. Last fall Denmark implemented a tax on saturated fats.

Do we have such taxes in this country?
There's not much political will in North America to impose any substantial taxes. What we've seen in the last two years is that in Colorado, Washington State and Washington, D.C., soda is no longer exempted from the sales tax. In Baltimore, there's a tax on soda, but it's fairly modest, and [the city is] using it to produce revenue, not necessarily to change behavior. There was a debate in New York state about a soda tax, and while some of it was about public health, the clear motivation [for the tax] was the state's fiscal situation in light of the economic downturn.

What has been learned about consumer behavior when a fat tax is in place?
Whenever something is more expensive, people tend to buy less of that item but end up buying other products that aren't necessarily any healthier. If you walk into a restaurant and your favorite hamburger dish costs a few dollars more, you're not likely to say, "I'll get a salad instead." You're more likely to get something else that's similar to the hamburger. If we tax colas, people might drink other sweetened beverages that aren't necessarily any better for them.

All studies suggest that for food in general, we're not particularly responsive to price. Food is a necessity. If I put a large tax on a food item, I can get people to buy less of it, but they will buy something else instead. If I raise the price of one brand of coffee, I can get people to buy another coffee brand. But if the price of all brands goes up, people will still buy coffee. Economists call this inelastic price response. In general, the demand for food is inelastic. When prices go up, we keep buying food, but we might reduce what we spend on other things.

Is a fat tax a political issue?
Any tax serves three purposes: It raises revenue, reallocates wealth—because someone pays more and someone pays less—and it changes behavior. Just because the motivation behind a tax might be to influence public health doesn't mean it doesn't do the other two things. We can't get away from doing all three with any tax. One thing I'd like to see in a public debate is a better understanding of these three things and honesty about what's motivating the tax.

"If you walk into a restaurant and your favorite hamburger dish costs a few dollars more, you're not likely to say, 'I'll get a salad instead.' You're more likely to get something else that's similar to the hamburger." —SEAN CASH

Who is affected most by fat taxes?
Such a tax can be regressive and fall more on households with lower incomes. The foods targeted often make up a larger percentage of the household budget for lower-income people because energy-dense, nutrition-poor foods are cheaper per calorie and are sometimes more available than healthier options. This would include many forms of candy, salty snacks and soft drinks.

Doesn't a higher tax on cigarettes help keep people from smoking?
Tobacco taxes are very high, and yet people still smoke. In Massachusetts, people pay $2.51 in state taxes and another $1.01 for the federal tax on top of the price per pack. It's hard to tell how much the higher cost made a difference in smoking rates, because the taxes were imposed at the same time there were public health campaigns and a move to put a stigma on smoking. So do the taxes help? I'm sure they do, but so do bans on smoking in bars, restaurants and public buildings.

Cigarettes come with warning labels. Would doing that for unhealthy foods make sense?
Using labels raises some interesting behavioral questions: Will people respond differently when they are told the price increase is for their health? If we put a big label on a food that reads, "We're taxing this because it's bad for you," it might have a bigger impact than just adding on a few cents. I was involved in a study where we found that people are less likely to choose a product with such a label, compared to one where we just raised the price.

Are there other ways to encourage healthy eating?
Some people have argued we should tax unhealthy food items to subsidize healthier items. I call that a "thin subsidy." It's expensive for taxpayers, but on the plus side, if we subsidize certain food items, such as fruits and vegetables, I worry less about people substituting unhealthy foods with other unhealthy foods, as with the fat tax.

Marjorie Howard, a senior writer in Tufts' Office of Publications, can be reached at marjorie.howard@tufts.edu.

A World of Good

Public health physician receives high honor from his native Australia by Linda Hall

IN HIS 35 YEARS FIGHTING MICRONUTRIENT malnutrition, Adjunct Professor Ian Darnton-Hill has witnessed the power of nutrition. He has seen the blindness, brain damage and fatal infectious diseases caused by chronic deficiencies in vitamin A, iodine, iron and zinc, and has seen how getting enough of such crucial micronutrients can save lives.

He has also watched nutrition come a long way as a career, a field of scientific inquiry and a global priority—a change from his early days when universities in his native Australia didn't even offer a full degree program in clinical nutrition.

That is why his recent appointment as an officer of the Order of Australia is so gratifying, less for personal reasons and more for the recognition it gives the field of international public health, a field he says he is proud to have served and lucky to have found.

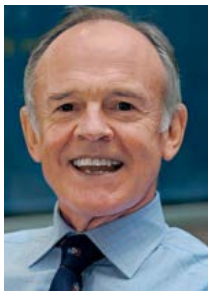
"I feel like I got away with a wonderful career," says Darnton-Hill, who has lectured and presented seminars at the Friedman School and cowritten papers with several faculty members over the years.

The honor, a preeminent way Australia recognizes achievement, cites his "distinguished service to the international community, particularly in the areas of public health and nutrition, to disease prevention and health promotion, and as a physician, academic and educator." Quentin Bryce, governor-general of Australia, has said honorees are "our truest guides to what matters most."

What has mattered most to Darnton-Hill, starting from his early days in medical school at the University of Adelaide, is a mixture of commitment and adventure, of practical outreach and scholarly discovery, of caring and curiosity.

A chance opportunity to be a medical exchange student in Indonesia first prompted new career horizons. He saw for the first time the impact of micronutrient malnutrition when he encountered a 2-year-old who was blind. "It really struck me that a simple thing—being vitamin-A deficient—could have such devastating effects," he says.

When it came time to do his residency, he set his sights on a road less traveled—perhaps never traveled—by requesting to go to Papua New Guinea. University administrators said no, but, with the help of a roommate who happened to be a lawyer, he was able to make the case that historical ties to the British Empire had established Goroka Base Hospital in Papua New Guinea with accredited training. To the dismay of university administration, Darnton-Hill was off on new adventures.



Ian Darnton-Hill

The highlands of Papua New Guinea had opened up to outsiders only about 40 years earlier, so Darnton-Hill's work there was very much outback outreach, including casualty response. "I remember we had to bring a patient back to the hospital in a light plane, a broken-off spear still in his chest," he says. "And there were my friends back in Adelaide taking blood pressure."

After completing traditional postgraduate work in the United Kingdom, focusing on anesthetics and family planning, Darnton-Hill, with his usual pluck, decided to stop by the World Health Organization in Geneva on his way home to Australia. He asked them what kind of doctors they needed. "Venereologists, toxicologists…the list went on, and then they said nutrition, and I said, 'Well, there you are.'"

There was no full degree program in clinical nutrition in Australia at that time, so he did coursework for a diploma in nutrition and dietetics, making him at the time the only medically qualified person in the country with a nutrition diploma.

Darnton-Hill's expansive résumé has included serving as director for Helen Keller International's vitamin A deficiency program in Bangladesh; World Health Organization medical officer/epidemiologist for noncommunicable diseases based in the Fiji Islands and later as a WHO regional adviser in nutrition for East Asia and the Pacific Island nations; and UNICEF's representative on a task force to accelerate progress toward the U.N. goal of dramatically reducing worldwide hunger by 2015.

"I think nutrition sometimes suffers from being everybody's business but no one's responsibility," he says. That is why some of the most gratifying work of his career has been helping to build global projects that put hunger and malnutrition at the top of stakeholders' priority lists.

As director of the Opportunities for Micronutrient Interventions Project (funded by the U.S. Agency for International Development), Darnton-Hill helped countries achieve goals defined by the first global conference devoted solely to the world's nutrition problems, convened in 1992 by the United Nations Food and Agriculture Organization and the WHO.

"It was one of the first times there was global consensus that something needed to be done about nutrition and [its role in] diseases," he says. "Most countries endorsed and developed a national plan of action for nutrition."

In 2008 Darnton-Hill cowrote one of a series of papers in *The Lancet* that presented evidence of the irreversible impacts of undernutrition on a child's development and highlighted the need for investment by donors and developing countries. The series served as a foundation for a current global initiative, called Scaling-Up Nutrition (SUN), in which governments and organizations are pooling resources to focus on the critical window between pregnancy and a child's second birthday.

For all the work he has done to help the disadvantaged, vulnerable and malnourished around the world, Darnton-Hill is the one who sounds grateful: It's been a "lucky" life, he says.

Everyone's a Comedian

Our sense of humor could be part of an evolutionary survival strategy by Taylor McNeil

Driving to work the other day, I heard an album title announced on the radio, *If It Weren't for Venetian Blinds, It'd Be Curtains for Us All*, and I burst out laughing. Why? The sheer silliness of it, the double meaning, hit me quickly, no doubt to the amusement of any driver who happened to see me cackling alone in the car.

We all have these moments—be it watching Homer Simpson, reading a New Yorker cartoon or sitting around the lunch table sharing quips with a sharp-witted coworker. Why we find some things funny and others not so much has been a staple for thinkers from Aristotle to Freud, who usually manage to kill the humor in anything they touch.

But there's a more fundamental question: Why do we have a funny bone in the first place? In 2004, after taking a psychology course on theories of humor, Matthew Hurley, A06, came up with a possible answer to that question. Humor, he speculated, could serve an evolutionary purpose. His thinking was that a big laugh might be our reward for vigilance in unmasking life's incongruities, a trait that can be critical for survival.

So he ambled into Daniel Dennett's office to propose the idea as a topic for his undergraduate honors thesis at Tufts. Intrigued, Dennett, the Austin B. Fletcher Professor of Philosophy and codirector of the Center for Cognitive Studies, agreed to be Hurley's adviser, along with Reginald Adams, now a psychology professor at Penn

ILLUSTRATION: BLAIR KELLY

State, who taught the course on humor.

Dennett found Hurley's resulting thesis so promising that he suggested trying to get it published. In the end, the student and his two professors collaborated on the book *Inside Jokes: Using Humor to Reverse-Engineer the Mind* (MIT Press, 2011).

The authors detail the multitudinous theories that have been offered over the centuries for how humor—or mirth, as it's called by academics—works. But most of those theories skirt the question that most intrigued Hurley, now a Ph.D. student at Indiana University.

"Why is there funny at all? I think that's the most important question we asked," Dennett says.

BRAIN CANDY

A lawyer was approached by Mephistopheles, who offered him a brilliant career as a defense attorney, leading to a seat on the Supreme Court and a Hollywood movie biopic—in exchange for the souls of his wife and three children. The lawyer thought and thought, sweat pouring off his brow. Finally, he looked up at Mephistopheles and said, "There's a catch, right?"

Human beings are anticipation machines: We're always making assumptions about what's coming next, usually based on very limited information. "Evolution gave us minds that tend to statistically fill in the blanks a lot—to make quick and dirty guesses as to what's going on around us, using experience as our guide," says Hurley, whose doctoral study is in cognition and emotion. Most of the time our minds do that work well, he notes, coming up with correct, or at least good enough, answers.

But occasionally we're just plain wrong. Hurley says that's where mirth plays a key role. The pleasure that a joke can bring motivates us to double-check our thoughts and ferret out our mistakes. "It's important to survival, and the only way it gets done is if it's fun," Dennett says. "That's the brain's way, Mother Nature's way, of getting us to do what it wants us to do."

A senior citizen is driving on the highway. His wife calls him on his cell phone and in a worried voice says, "Herman, be careful! I just heard on the radio that there is a madman driving the wrong way on Route 280!"

Herman says, "Not just one, there are hundreds!"

We catch the error—we'd been expecting one thing, and when another pops up instead, it strikes us as funny.

Yet our brains, say Hurley and Dennett, have to perform many functions—and looking for incongruities is just one. So while the main purpose of humor is to help us survive, the enjoyment it provides has taken on a broader significance in our lives. "In the same way that chocolate cake is a supernormal stimulus for a sweet tooth—nothing that nature intended—so humor is chocolate cake for the funny bone," Dennett says.

Texan: "Where are you from?"

Harvard grad: "I come from a place where we do not end our sentences with prepositions."

Texan: "Okay—where are you from, jackass?"

The evolutionary theory of humor isn't exactly easy to prove, but Hurley says the "cognitive-mechanical details" of it can be put to the test by psychology researchers. Neuroscientists have been studying brain activation in humorous circumstances, he notes, and they've also been "finding support for the probably-not-too-controversial idea of reward system activation," one of the mechanisms by which the authors say humor works.

SOCIAL TALENT

I was wondering why the Frisbee was getting bigger, and then it hit me.

Humor isn't just about having a good yuk at our own expense, or someone else's. It's a vital social skill, Dennett says. "Humor is part of human intelligence," he explains. "There's a lot of social talent, social competence, which is manifest in a sense of humor, and if you don't have a sense of humor, it's not like being tone deaf. Not having a sense of humor is a severe disability."

The pleasure that a joke can bring motivates us to double-check our thoughts and ferret out our mistakes. "That's the brain's way, Mother Nature's way, of getting us to do what it wants us to do."

—Daniel Dennett

For example, last summer, when Herman Cain was still in the Republican presidential primary, he addressed a large gathering of conservatives, winning them over easily with jokes told with the timing of a stand-up comic. But when Mitt Romney came on stage next, his jokes fell flat; it was clear that his connection with the audience was weak at best.

Ad in a newspaper: "Illiterate? Write today for free help."

Another interesting point is that we seem to be the only species with a sense of humor. Our close relatives, the bonobos and chimpanzees, apparently appreciate fun, but it looks very different from humor. Again, Dennett says, that fits with the evolutionary role of humor: It's an attribute that would have evolved as humans evolved.

There is, of course, an occupational hazard of studying humor: You lose your sense of it. It's what's called being "joke blind," like eating so many sweet things that you can't tell what's sweet anymore.

A hole has been found in the nudist camp wall. The police are looking into it.

Nevertheless, Hurley says he himself is as full of mirth as ever. All the analyzing "killed many a joke," he says, "but new humorous things happen every day, and I think I'm still as sensitive to them as I always was."

Taylor McNeil, the senior news editor in Tufts' Office of Publications, can be reached at taylor.mcneil@tufts.edu.

Turning Around an Epidemic

Philanthropy fuels a strategy to prevent childhood obesity
by Mark Sullivan

As an athlete, Peter Dolan, A78, A08P, has run marathons, competed in triathlons and completed the grueling Hawaii Ironman challenge. As a philanthropist, he is tackling an even bigger challenge: childhood obesity.

A Tufts University trustee and former CEO of Bristol-Myers Squibb, Dolan and his wife, Katie, have donated $1 million to a national initiative known as ChildObesity180, which he is chairing at the Friedman School.

ChildObesity180 (www.childobesity180.org) seeks to become a major catalyst for prioritizing and driving the necessary systemic changes to reverse the trend of childhood obesity within a generation. Founded in 2009, ChildObesity180 draws on the expertise and reach of senior decision-makers from the highest levels of government, academia, public health advocacy, community organizations, the food industry and the media to drive an integrated national strategy to prevent childhood obesity.

With this new donation, Peter and Katie Dolan have made nearly $2.7 million in gifts and pledges to Tufts University in support of financial aid, the Summer Scholars undergraduate research program, the Tufts Marathon Challenge, the School of Medicine and athletics. A previous million-dollar donation established the Dolan Family Endowed Scholarship Fund.

The Dolans' latest gift supports the fundraising campaign under way for this large-scale campaign to make our kids healthier. ChildObesity180 was publicly launched last fall with a $6.9 million grant from the Robert Wood Johnson Foundation, and $14.6 million in total funding has been raised so far. "This epidemic is too important to wait another moment," says Christina Economos, Ph.D., N96, the Friedman School's New Balance Chair in Childhood Nutrition, who is vice chair and director of ChildObesity180.

"Childhood obesity is the preeminent public health issue of our time," she says. "Today, one-third of children in America are overweight or obese and on track to experience catastrophic health conditions, swamp health-care budgets and create unprecedented challenges across society."

The Dolan gift will provide core support for ChildObesity180 and serve as an engine to advance the group's work, says the project's codirector, Miriam Nelson, Ph.D., N85, N87, professor and director of the John Hancock Research Center on Physical Activity, Nutrition and Obesity Prevention at the Friedman School. "Peter is an amazing champion and leader for ChildObesity180," she adds.

Robin Kanarek, Ph.D., interim dean of the Friedman School, notes: "Without Peter's vision and leadership, ChildObesity180 would not be possible. We are proud to be working with him to reverse the obesity epidemic in this country."

ChildObesity180 takes a high-level view of all activities in the childhood obesity field and analyzes them to determine

PHOTO: CORBIS

the best, most actionable and impactful opportunities. Through its unique decision-making matrix, the leadership of ChildObesity180 reviewed a comprehensive set of evidence-based obesity prevention recommendations and identified four priority areas on which to focus: children's access to healthier foods, physical activity, marketing to children and eating out in restaurants.

To address these strategic areas and promote a collective impact, ChildObesity180 is developing and executing a portfolio of initiatives designed to affect numerous aspects of a child's daily environment. This past February, with help from First Lady Michelle Obama, ChildObesity180 launched a nationwide competition for innovation in school-based physical activity as part of its Active Schools Acceleration Project.

"We know there's so much good work going on all across this country to get our kids up and moving every single day," Obama said in a video announcing the Active Schools Acceleration Project Competition, which ran through April. "So we want to find the best school programs and technology ideas that increase physical activity for kids—and then help them reach even more children throughout America."

In the spring, the innovation competition—supported by more than $1.2 million from 13 of the nation's leading health-insurance companies and foundations—awarded $500,000 in prizes, with individual awards of up to $100,000, for the most creative, impactful and scalable programs and technological innovations that promote quality physical activity for children during the school day. The next step will be for the Active Schools Acceleration Project to replicate the winning models on a larger scale, with the goal of achieving sustainable quality physical activity in schools.

The Active Schools Acceleration Project was the second initiative launched by ChildObesity180. The first, Healthy Kids Out of School, is a collaboration among nine of the nation's largest after-school, sports and extracurricular organizations. Convened by ChildObesity180, the leaders of these groups developed and adopted universal nutrition and physical activity principles from a broad list of evidence-based recommendations for combating childhood obesity. Detailed implementation plans are under way, and a three-year, $1.5 million grant from the Harvard Pilgrim Health Care Foundation will help catalyze these efforts.

Through its portfolio of high-impact, evidence-based initiatives and its commitment to research and evaluation, ChildObesity180 will continue to bring partners to the table to work together in pursuit of a common mission: reversing the trend of childhood obesity within a generation, says Economos.

For more information about ChildObesity180, including how you can support its efforts, contact Cindy Briggs Tobin, senior director of development and alumni relations at the Friedman School, at 617.636.2940 or cindy.briggs@tufts.edu

CHILDOBESITY180 DONORS

(as of May 1, 2012)

Core Support

Robert Wood Johnson Foundation
JPB Foundation
Peter and Katherine Dolan

Healthy Kids Out of School

Harvard Pilgrim Health Care Inc.

Active Schools Acceleration Project (ASAP)

Blue Cross and Blue Shield of Florida Foundation
Blue Cross and Blue Shield of North Carolina
Blue Shield of California
CIGNA Foundation
EmblemHealth
Health Alliance Plan
HealthPartners, Inc.
Horizon Blue Cross Blue Shield of New Jersey
Humana Inc.
Kaiser Permanente
The Regence Group
Tufts Health Plan
Wellpoint Foundation

WHY THEY GIVE

"We had been seeking a link with an academic institution. With its interdisciplinary focus on nutrition, agriculture and policy, the Friedman School seemed a very good match. We felt we could make a difference here."

—Barbara Rose, executive director, Aid for Africa, which established an endowed fund to support student research in Africa designed to improve food security and reduce poverty

Setting a Personal Compass

88 new graduates join the ranks of Friedman School alumni

Looking out at the packed Cohen Auditorium at the Friedman School's 31st commencement ceremony on May 20, Interim Dean Robin Kanarek said the scene was a far cry from Tufts' first nutrition school commencement, when only a handful of degrees were awarded. This year's ceremony would see 78 master's degrees and 10 doctoral degrees handed out.

Peter Dolan, A78, A08P, vice chair of the Tufts University Board of Trustees, offered one specific piece of advice in his commencement address: take the time to write a mission statement for your life.

"I can't help you define your North Star or create your personal compass," he said. "But I really do conclude that having one makes all the difference."

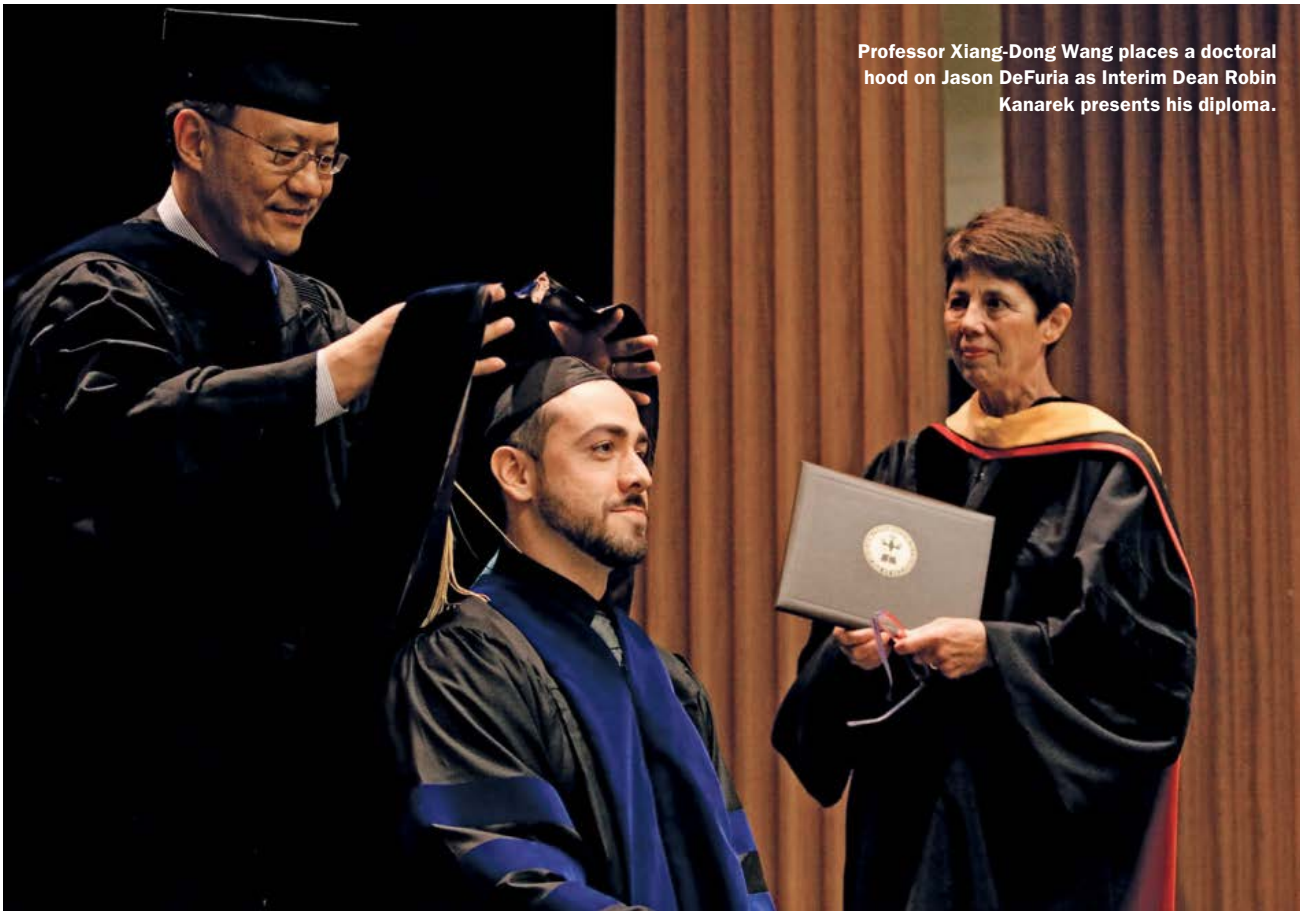
He said he wrote his first personal mission statement in 1985 as part of a leadership seminar to which his employer at the time, General Foods, sent him. The statement covered professional and personal priorities.

Ironically, one of the first things he did as a result of his mission statement was to leave the company—it didn't fit with his newly minted goals.

"I still have the original version of what I wrote, have referred to it often and believe it guided me at key decision points of the last 25 years," he said.

Dolan went on to become chairman and CEO of Bristol-Myers Squibb. When he left the company after 18 years, he revised his mission statement to focus on health and wellness, which led him, in part, to become director of Vitality Health, a for-profit health and wellness company, and to chair ChildObesity180, a team of leaders and experts, centered at Tufts, working to reverse the nation's child obesity crisis. (See related story, page 34.)

"Don't be afraid to think big and visualize that ideal trajectory, just like the athlete who sees that perfect shot," he told the Friedman School's newest alumni.



Professor Xiang-Dong Wang places a doctoral hood on Jason DeFuria as Interim Dean Robin Kanarek presents his diploma.

PHOTO: EMILY ZILM

Left, alumni, friends and family at the reunion brunch. Above, Karen Weikel, N08, N11, received the Innovation/Up and Coming Award.

Above, Xiang Gao, N05, received the Leadership and Expertise Award. Right, Alicia Romano, N10, Grace Phelan, N05, and Kerri Hawkins, N06

Randa Wilkinson-Bouvier, N85, center, who received the Leah Horowitz Humanitarian Award, was congratulated by Alexis Felder and Monique Sternin.

REUNION 2012

In April, the Friedman School's 10th annual All-Alumni Reunion Weekend celebration brought alumni from around the world back to campus for three days of events, including a career panel with Agriculture, Food and Environment Program graduates; a volunteer opportunity at the Greater Boston Food Bank; and the annual Gershoff Symposium, titled "Food Supply and Demand: Who's in Control?" The brunch featured a keynote address from Theresa Marquez, chief mission officer at Organic Valley, and honored the 2012 Alumni Award winners, nominated by their peers for their outstanding contributions to nutrition.

PHOTOS: MATTHEW MODOONO

Can We Hear You Now?

THE FRIEDMAN SCHOOL ALUMNI Association is here for you—and we want to hear from you! Over the past year the school has hosted alumni events in San Diego, Boston, Atlanta, Phoenix, Washington, D.C., and Florida. We have also expanded our online efforts so you can be connected from anywhere in the world. You can join us on Wednesdays for the live streaming of the Friedman Weekly Seminar Series; a full schedule can be found at nutrition.tufts.edu/event/friedmanseminar. In addition, we held four webinars with Robin Kanarek, Jennifer Hashley, Miriam Nelson and William Masters. These and many others great online events are archived for your convenience at nutrition.tufts.edu/event/recorded.

But what we really want to know is, what do you want?

The Alumni Association strives to create an engaged community of alumni around the world, but we need your input to make this happen.

DO YOU WANT TO...

Host an alumni event at your home or business?

Meet with students on your trip through Boston?

Post/search job opportunities for the Friedman School community?

Receive more nutrition research updates?

Visit *alumni.nutrition.tufts.edu*, join us on Facebook and LinkedIn and tell us what you want and need from *your* Alumni Association. Do not hesitate to send me an email at ausen@alumni.tufts.edu with your thoughts and suggestions. We are only successful with your involvement. Hope to hear from you soon!

Sincerely,

ABBY USEN BERNER, N03

PRESIDENT, FRIEDMAN SCHOOL ALUMNI ASSOCIATION

P.S. Don't forget to visit www.tuftsalumni.org and update your contact information. We can't keep you up to date on school happenings and events without your current address, email and phone.

Class Notes

G75 **Helene Fuchs** led a Networking 101 workshop in April for current Friedman School students on the Boston campus. She is president of Helene Fuchs Associates (www.helenefuchsassociates.com), which collaborates with nonprofit, for-profit, private and public health-care organizations to develop and improve programs, products and services, and facilitates adaptations to health-care reform initiatives and market changes.

G89 **Diane McKay**, **N97**, **N00**, is a 2012 recipient of a Tufts Innovates! seed grant. This program, introduced by the Office of the Provost in December 2010, facilitates the exploration of new approaches to enrich student learning. McKay, along with Friedman School colleagues Kelly Kane and Patrick Connell, received funding for their project "Nutrition 101: Using Blended Learning and Educational Technology to Improve Student Learning in a Large-Enrollment Course."

N82 **Beverly Tepper**, **N86**, and **Josh Miller**, **N90**, **N93**, contributed to Interim Dean Robin Kanarek's most recent book, *Diet, Brain and Behavior: Practical Implications* (CRC Press, 2012), which she co-edited with Harris R. Lieberman.

N85 **Randa Wilkinson-Bouvier**, director of training for the Positive Deviance Initiative, is the 2012 recipient of the Leah Horowitz Humanitarian Award given by the Friedman School Alumni Association.

N87 **Tom Hughes**, **A10P**, president and chief executive at Zafgen, gave a Friedman Weekly Seminar titled "Obesity Pharmacotherapy: Past, Present, Future" on April 18. All Friedman Seminars can be viewed at http://nutrition.tufts.edu/event/recorded.

N90 **Josh Miller**, see N82.

N93 **Roger Fielding's** research on sarcopenia was featured in *The Boston Globe* article, "Stopping Age-related Muscle Loss: Put the Brakes on the Loss with Strategic Exercise and Protein Intake."

N97 **Susan Cooper Greeley** just published her first cookbook, which is also a diet book. It's called *Lighten Up!* and is part of the Cooking with Trader Joe's cookbook series. It's now available in stores and online at amazon.com and barnesandnoble.com.

N99 **Kate Houston**, director of federal government relations for Cargill, spoke during Professor James Tillotson's Health Claims class in April.

N00 **Andrew Shao**, vice president for global product science and safety

at Herbalife, gave a Friedman Weekly Seminar titled "Scientific, Policy, Regulatory and Market Challenges: The Perspective of a Global Company" on March 28. All Friedman Seminars can be viewed at http://nutrition.tufts.edu/event/recorded.

N01

Helen Costello received the 2012 Andrew L. Felker Award from the New Hampshire Department of Agriculture, Markets and Food in recognition of her contributions to the health and vitality of New Hampshire.

N02

Kimberly Dong, see N08.

Tara Mardigan gave the keynote address at the Tufts Active Citizens Boston Gala in February. The event helped raise funds for Friedman School Student Council community outreach activities and Future Chefs.

N04

Katie Cavuto Boyle was the keynote speaker for the 2012 Massachusetts Student Dietetic Association career development workshop held at the Jean Mayer USDA Human Nutrition Research Center on Aging on April 20.

N05

Sonya Elder, **N09**, food services director for the Brookline (Mass.) Public Schools, was named 2012 Brookline Woman of the Year by the Brookline Commission for Women, in recognition of her efforts to get students to eat healthier. She introduced vegetarian options and more fruits and vegetables on school lunch menus. "A lot of people think of school food as being highly processed," Elder said. "If you look at our menus, you'll see a lot of fresh produce and raw ingredients. We're trying to make it more like home cooking."

Xiang Gao, an assistant professor of medicine at Harvard Medical School and an associate epidemiologist at Brigham and Women's Hospital, is the 2012 recipient of the Leadership and Expertise Award given by the Friedman School Alumni Association.

Stacey King married Shamsher Bam on September 25, 2011. She is enjoying her new role at the Cambridge Public Health Department as director of its Community Health & Wellness Division.

Grace Phelan and **Sarah Sliwa**, **N13**, organized an "Entrepreneurial Alumni" career panel in March. It featured **Nicki Briggs**, **N08**, director of communications for Chobani Yogurt; **Jessica Jones-Hughes**, **N09**, vice president of Oké USA Fair Trade Fruit Company; and **Amy Scheuerman**, **N10**, assistant editor for new media at *America's Test Kitchen*.

Gabrielle Serra just started a new job as a senior policy associate with the Public Health Institute in Washington, D.C.

N06

Britt Lundgren, director of organic and sustainable agriculture at Stonyfield Farms, gave a Friedman Weekly Seminar titled "The Case for Labeling Genetically Engineered Food" on February 15. All Friedman Seminars can be viewed at http://nutrition.tufts.edu/event/recorded.

N07

Janel Funk, see N08.

N08

Dustin Burnett moderated the career panel at the 2012 Massachusetts Student Dietetic Association career development workshop. It included fellow alumni **Nicki Briggs**, **Kimberly Dong**, **N02**, and **Janel Funk**, **N07**.

Shirley Y. Chao, director of nutrition of the Massachusetts Executive Office of Elder Affairs, has been nominated to the board of directors and as treasurer of the National Association of Nutrition and Aging Services Programs, which provides older adults with healthful food and nutrition through community-based services. She was also elected as the chair of the Healthy Aging Dietetic Practice Group of the Academy of Nutrition and Dietetics for 2012–13.

N10

On September 24, 2011, **Melissa Bailey**, **V03**, and her husband, John, welcomed a baby boy, John Hayden Bailey. Melissa is keeping busy with baby and her position as standards director for the National Organic Program at the USDA.

Ashley Reynolds is working in public relations at the agency Mullen for a variety of food and nutrition clients.

Amy Scheuerman, see N05.

Dawn Undurraga writes, "We welcomed our son, Linus Undurraga, into the world on October 13, 2011, at 1:05 p.m. He was a robust 8 pounds and 11 ounces."

N11

Shannon Washington is a scientist II food product developer for Ralcorp Holdings Inc.'s Ralcorp Snacks, Sauce and Spreads Division.

Karen Weikel is the 2012 recipient of the Innovation/Up and Coming Award given by the Friedman School Alumni Association.

Lisa Gross, G11, center left, and Maura Schorr Beaufait, N10, MPH10, center right

Branching Out

The way Maura Schorr Beaufait, N10, MPH10, describes it, she has always been a practical, "draw-within-the-lines" sort of person. In her job as coordinator of healthy food access initiatives at the Bowdoin Street Health Center in Boston's Dorchester neighborhood, her work has been focused on "tangible outcomes," such as getting healthier foods into corner stores and getting more folks to shop at the local farmers market.

That's why she finds her other role, as director of operations for the Boston Tree Party, a little intimidating. Last year the public art project, conceived by artist Lisa Gross, G11, brought together groups from across the city to plant 35 pairs of three-foot heirloom apple trees throughout Boston in a symbolic, festive commitment to environmental and community health. The party's inaugural event celebrating the birth of an urban orchard was a freewheeling blend of public art, urban agriculture and civic engagement, complete with colorful banners, a brass band and soaring rhetoric.

"It's exciting for me to be engaged in a project that is so open-ended and artistic and creative, because that's not the way I view myself or generally operate," Beaufait says. "It really challenges me to think about what's possible—not just riffing on someone else's theme, but something entirely new."

As the group was gearing up for its second round of tree plantings this spring, Beaufait is still the most grounded one on the team, always asking such things as, "But where is the money going to come from?" She discovered that funding art is very different from the outcome-centered grant-writing process she is used to. "The funders we have say, 'I see power in the seeds of your ideas. I trust you.' "

Of course, the Boston Tree Party is not purely symbolic. Once they start producing, the trees will be an outlet for free, fresh, healthy food for their communities, to the tune of 10,000 to 15,000 apples every year. It is just a small part of the civic fruit that Beaufait, Gross and the others hope to harvest from the experiment.

PHOTO: KELVIN MA

Substituting whole grains for refined ones decreases visceral fat.*

Research shows that hibiscus tea can lower your blood pressure.*

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Oats may reduce the inflammation that leads to cardiovascular disease.*

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*This information has appeared in *Tufts Nutrition* magazine and represents the research of our community.

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NOT MY REVOLUTION

Food writer Michael Pollan has advised people not to eat anything their great-grandmothers wouldn't recognize as food. But what if your great-grandmother was a slave? The call to eat local and organic may be well intentioned, says Julian Agyeman, professor and chair of urban and environmental policy and planning in Tufts' School of Arts and Sciences, but the message doesn't resonate much outside of white, middle-class society. For more on the story, turn to page 26.

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