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Lustig, Robert H

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buy previously unaffordable luxuries for her grandson including toiletries, fresh produce, winter blankets, and a nightlight.

Like Beatrice, single women making less than \$10 000 yearly headed many of the families we served. The good news for these women and their children was 2-fold. First, these types of families are the EITC's target population and, as such, received thousands of dollars in tax credit. Second, for those who filed in prior years, StreetCred saved them money previously lost to the for-profit tax-filing industry by providing free services in the comfort of a trusted setting: their pediatrician's office. These women expressed both gratitude and practical impact as they reported their ability to not only more fully meet their children's basic needs, but also take a first step toward financial stability by paying off loans, which are stories consistent with national data on the ways taxpayers use EITC monies.⁷ Moreover, StreetCred empowered families with EITC education. Some clients expressed enthusiasm about working more hours in the coming year to qualify for a larger EITC on their next tax return. Other single mothers learned that they, not their ex-

husbands, should claim the children as tax-return dependents because they served as primary caretakers.

StreetCred is another example of how the US health care system can be a gateway to the public benefits, community resources, and financial stability supporting low-income parents, like Beatrice, in one of the most important jobs in the United States: raising healthy children. The trust and regular contact between families and their children's medical professionals present a special opportunity to screen for and address the social determinants of health. StreetCred aims to make accessing programs, such as the EITC, cheaper, faster, and easier to understand. The program's short-term goals are 2-fold: (1) expand free tax-preparation services to other clinics and hospitals serving low-income families with children and (2) expand its services portfolio with additional asset-building tools so families might truly break the cycle of poverty. Innovative programs extending beyond the traditional boundaries of pediatric health services remain an important approach to promoting the health and development of our patients growing up poor.

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REFERENCES

1. Center on Budget and Policy Priorities. Policy basics: the earned income tax credit. <http://www.cbpp.org/research/federal-tax/policy-basics-the>

-earned-income-tax-credit. Published January 15, 2016. Accessed July 7, 2016.

2. Marr C, Huang C-C, Sherman A, Debot B. EITC and child tax credit promote work, reduce poverty, and support children's development, research finds. Center on Budget and Policy Priorities website. <http://www.cbpp.org/research/federal-tax/eitc-and-child-tax-credit-promote-work-reduce-poverty-and-support-childrens>. Published October 1, 2015. Accessed July 7, 2016.

3. US Internal Revenue Service. About EITC. <http://www.irs.gov/EITC-Central/abouteitc>. Accessed April 1, 2015.

4. Weinstein P, Patten B. *The Price of Paying Taxes II: How Paid Tax Preparer Fees are Diminishing the Earned Income Tax Credit (EITC)*. Washington, DC: The Progressive Policy Institute; 2016.

5. Council on Community Pediatrics. Poverty and child health in the United States. *Pediatrics*. 2016; 137(4):e20160339.

6. Holzer HJ, Shanzenbach DW, Duncan GJ, Ludwig J; National Poverty Center. National Poverty Center Working Paper Series, 07-04: The economic costs of poverty in the United States: subsequent effects of children growing up poor. http://www.npc.umich.edu/publications/u/working_paper07-04.pdf. Published January 2007. Accessed July 7, 2016.

7. Goodman-Bacon A, McGranahan L. How do EITC recipients spend their refunds? *Econ Perspect*. 2008;32(2):17-32.

VIEWPOINT

Processed Food—An Experiment That Failed

Robert H. Lustig, MD, MSL

Department of Pediatrics, University of California, San Francisco; and Philip R. Lee Institute for Health Policy Studies, University of California, San Francisco.

Corresponding

Author: Robert H. Lustig, MD, MSL, Division of Pediatric Endocrinology, University of California, San Francisco, 550 16th St, PO Box 0434, San Francisco, CA 94143 (rlustig@ucsf.edu).

Those of us who have participated in science know that 9 of every 10 experiments are failures. Now imagine that the last 50 years has been a grand clinical research experiment, with the American population as unwitting participants, conducted by 10 principal investigators—Coca-Cola, PepsiCo, Kraft, Unilever, General Mills, Nestlé, Mars, Kellogg, Proctor & Gamble, and Johnson & Johnson. In 1965, these corporations posed the hypothesis that processed food is better than real food. To determine if the experiment was a success or a failure, we have to examine the outcome variables. In this case, there are 4: food consumption, health/disease, environment, and cash flow, divided into companies, consumers, and society.

Processed food is defined by 7 food engineering criteria; it is mass produced, is consistent batch to batch, is consistent country to country, uses specialized ingredients from specialized companies, consists of prefrozen macronutrients, stays emulsified, and has long shelf life or freezer life.¹

Furthermore, 11 nutritional properties distinguish processed food.² (1) Too little fiber. When fiber (soluble and insoluble) is consumed within food, it forms a gelatinous barrier along the intestinal wall. This delays the intestine's ability to absorb nutrients, instead feeding the gut microbiome. Attenuation of the glucose rise results in insulin reduction. Attenuation of fructose absorption reduces liver fat accumulation. (2) and (3) Too few ω -3 and too many ω -6 fatty acids. ω -3s are precursors to docahexaenoic and eicosapentanoic acids (anti-inflammatory). Conversely, ω -6s are precursors of arachidonic acid (proinflammatory). Our ratio of ω -6 to ω -3 fatty acids should be approximately 1:1. Currently, our ratio is about 25:1, favoring a proinflammatory state, which can drive oxidative stress and cell damage. (4) Too few micronutrients. Antioxidants, such as vitamins C and E, quench oxygen radicals in peroxisomes to prevent cellular damage, while others, such as carotenoids and α -lipoic acid, prevent lipid peroxidation. (5) Too many

trans-fats. These fats cannot be oxidized by mitochondria owing to the trans-double bond, so they line arteries and the liver and generate oxygen radicals. Of note, the US Food and Drug Administration declared in 2013 that trans-fats are not “generally recognized as safe,”³ so they should soon disappear from the food supply. (6) Too many branched-chain amino acids. Valine, leucine, and isoleucine are essential amino acids required for muscle biosynthesis. But when consumed in excess, they are deaminated in the liver and diverted to de novo lipogenesis, which increases liver fat. (7) Too many emulsifiers. Emulsifiers keep fat and water (eg, ice cream or lasagna) from separating. However, emulsifiers are detergents and may strip away the mucin layer that protects intestinal epithelial cells, predisposing individuals to intestinal disease or food allergy. (8) Too many nitrates. Nitrates (cured meat) can be metabolized into nitrosoureas, which can predispose individuals to colon cancer. (9) Too much salt. Approximately 15% of the population is salt sensitive and can manifest with hypertension and cardiac disease. (10) Too much ethanol. Ethanol is converted into liver fat and drives oxidative stress. While clearly a concern in adults, it is less likely that ethanol poses a metabolic risk in most children, as their access is limited. (11) Too much fructose. Children consume fructose instead. In fact, fructose is metabolized by de novo lipogenesis in the liver exactly like ethanol. Indeed, sugar (ie, sucrose and high-fructose corn syrup) is the “alcohol of the child,”⁴ which is why children now get the diseases of alcohol consumption (eg, type 2 diabetes, dyslipidemia, and nonalcoholic fatty liver disease) without consuming alcohol. Furthermore, 74% of all the items in the grocery store contain added sugar⁵; this makes sugar the marker for processed food.

Let's assess each of the 4 outcome measures in turn. First is food consumption. The United States spends only 7% of gross domestic product on food, allowing us, the most obese nation, to buy more. There's no question that food consumption is way up—an increase in 187 kcal/d in men, 335 kcal/d in women, and 275 kcal/d in teens since 1995. But what are these calories? Not fat, the amount of which has stayed stable. The increase is in refined carbohydrates, half of which are sugar. In the last 30 years, while meat has declined from 31% to 21% of food dollars, processed foods and sweets have increased from 11.6% to 22.9%.

Next is health/disease. There's no question both obesity and type 2 diabetes have increased astronomically. Sugar consumption predicts metabolic syndrome in adolescents, regardless of calories or body mass index. When we substituted starch for sugar in children, their metabolic syndrome resolved.⁶ In fact, research shows that sugar is a proximate cause of type 2 diabetes, dyslipidemia, and nonalcoholic fatty liver disease.⁷

Third is environment. The World Wildlife Federation argues that production of sugar-related crops leads to soil erosion and an annual loss of 6 million hectares of arable land. We certainly see this in the Everglades and the Amazon. Furthermore, crop monoculture (ie, corn and soy) to produce processed food has led to increased atrazine use, increased nitrate contamination, the development of herbicide resistance, and the appearance of “superweeds.”⁸

And lastly, cash flow. Until 2012, the processed food, sugar, and beverage companies fared better than the rest of the Standard and Poor 500; however, since 2013, their market performance has been suboptimal, highlighted by the firing of 1800 Coca-Cola employees in 2014 to save \$3 billion and the firing of McDonald's CEO Don Thompson. For consumers, processed food costs half as much per calorie as real food, and its trajectory of increase over time is lower; this would ostensibly make processed food a better short-term deal. However, the money spent on insurance premiums, the reduction in years of work due to disability, and the increase in years of life lost due to chronic disease over the long term more than eclipses the savings to consumers. Health care has grown from 2% in 1965 to 17.9% in 2014 of gross domestic product and is estimated to reach 21% by 2020. Currently, the food industry grosses \$1.46 trillion annually, of which 45%, or \$657 billion, is gross profit. However, health care costs \$3.2 trillion annually, of which 75% are spent on the diseases of metabolic syndrome; 75% of metabolic syndrome costs could be prevented if we changed our collective diet. That adds up to \$1.8 trillion dollars wasted; we lose triple what the food industry makes. This is unsustainable. Obamacare cannot stem the tide because there's no prevention to long-term disease other than changing the diet. This is why Morgan Stanley predicted 0.0% economic growth by 2035 based on our current high-sugar model⁹ and why Credit Suisse called for taxation of sugar to limit the obesity and diabetes crises.¹⁰ (Thus far, public referenda have passed in Berkeley, San Francisco, Oakland, and Albany, California; Boulder, Colorado; Cook County, Illinois; and Philadelphia, Pennsylvania.)

Given these outcomes, the conclusion is clear: processed food is an experiment that failed. Processed food is high in sugar and low in fiber. There's only one recourse—real food, which is low in sugar and high in fiber. Real food is what the world ate for millennia without risk of long-term disease. But that's not what the 10 biggest food corporations are selling. One-third of American mothers today don't even know what real food is or how to cook; they and their children are destined to remain hostages to the processed food industry. Pediatricians provide anticipatory guidance. Dispelling the processed food myth must be priority number 1.

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REFERENCES

- Blythman J. *Swallow This: Serving Up the Food Industry's Darkest Secrets*. London, England: Fourth Estate; 2015.
- Lustig RH. Metabolic syndrome and the “Western Diet”: science and politics. In: Kiess WWM, Maffei C, Sharma A, eds. *Metabolic Syndrome and Obesity in Childhood and Adolescence*. Vol 19. Basel, Switzerland: Karger Publishers; 2015: 136-146.
- US Food and Drug Administration. The FDA takes step to remove artificial trans fats in processed foods. <http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm451237.htm>. Accessed June 16, 2015.
- Lustig RH. Fructose: it's “alcohol without the buzz.” *Adv Nutr*. 2013;4(2):226-235.
- Ng SW, Slining MM, Popkin BM. Use of caloric and noncaloric sweeteners in US consumer packaged foods, 2005-2009. *J Acad Nutr Diet*. 2012;112(11):1828-34.e1-6.
- Lustig RH, Mulligan K, Noworolski SM, et al. Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome. *Obesity (Silver Spring)*. 2016; 24(2):453-460.
- Lustig RH. Sickeningly sweet: does sugar cause diabetes? yes. *Can J Diabetes*. 2016;40(4):282-286.
- Gurian-Sherman D, Mellon M. The rise of superweeds—and what to do about it. http://www.ucsus.org/sites/default/files/legacy/assets/documents/food_and_agriculture/rise-of-superweeds.pdf. Accessed December 15, 2016.

9. Morgan Stanley Research. The bittersweet aftertaste of sugar. <http://static.la Tribune.fr/463077/etude-morgan-stanley-impact-diabete-sur-l-economie-mondiale.pdf>. Accessed December 15, 2016.

10. Credit Suisse Research Institute. Sugar: consumption at a crossroads. http://wphna.org/wp-content/uploads/2014/01/13-09_Credit_Suisse_Sugar_crossroads.pdf. Accessed December 15, 2016.

VIEWPOINT

In the Aftermath of the National Children's Study Is Large Birth Cohort Data Still a Priority?

Terence Dwyer, MPH, MD, MB, BS, AO

Nuffield Department of Obstetrics and Gynecology, The George Institute for Global Health, University of Oxford, Oxford, United Kingdom.

Per Magnus, MD, PhD

Norwegian Institute of Public Health, Oslo, Norway.

Jørn Olsen, MD, PhD

The Department of Clinical Epidemiology, Aarhus University Hospital, Aarhus, Denmark.

The 2014 decision to stop the US National Children's Study (NCS)¹ brings to the forefront questions about what has been lost and how studies such as this might still be important almost 20 years after initiation in 2000. The rationale then was clear.² Little progress had been made in the previous decades in understanding the causes of many major childhood disorders, and there was insufficient evidence available to confidently mount interventions to prevent many of them. A lack of evidence from cohort studies with prospective data had left a major evidence gap in childhood disease etiology, in stark contrast to efforts involving successful research on adult diseases where cohort studies were a central component.

Important pediatric conditions for which prospective data might be critical included birth defects, childhood cancer, type 1 diabetes, and autism.² The exposures of interest for these conditions embraced infections of the mother and infant, environmental chemicals, nutrition, and growth of the fetus. Environmental factors, such as infections and many chemicals, could not be measured validly with retrospective data because parents' recall of these had been shown to be affected by bias.^{3,4} Analysis of biospecimens collected after disease onset failed to provide data on what was happening during the key time when disease was evolving. Therefore, the field of pediatric disease prevention could not move forward without prospective data of this kind.

The barrier had been the cost and effort required to conduct cohort studies of the size needed to provide adequate power to investigate important pediatric diseases that, relative to adult diseases, are uncommon. Only very large cohort studies, with at least 100 000 participants, would be sufficiently powered to have a chance of providing reliable evidence of an association with potential risk factors on the less common serious diseases of interest.

Nonetheless, the proposition that this obstacle must be overcome if progress was to be made in understanding pediatric disease causation was clearly gaining strong support toward the end of the 20th century. As well as the decision to start the NCS, Danish and Norwegian birth cohort studies were launched in the period leading up to 2000. They benefited from the comprehensive sampling frames for health studies and health data linkage that those nations possess. Partly as a consequence of

this, they were able to enroll and measure mothers in pregnancy and their infants and collect data on their 100 000 participants for what they perceived as acceptable costs, around US\$20 million.

The NCS faced bigger cost hurdles because of the lack of such infrastructure. In addition, its planners argued that prenatal recruitment was necessary to avoid potential selection bias associated with failure to include mothers who miscarried or experienced a stillbirth and decided to sample from the only feasible sampling base in the United States that would include all potential new mothers: the household. Importantly, this would have enabled home-based collection of exposure data that the other more modestly funded international cohorts were unable to incorporate. Analyses conducted later by the NCS team revealed that this sampling approach would inflate the costs over an alternative scenario involving sampling of prenatal clinicians and birth hospitals by a ratio of 30:1. However, it seems that the lower-cost approach was not seen as sufficiently attractive an option to salvage the study in the minds of those who undertook the final review. It has been reported that when the NCS was stopped, it had already cost more than \$1 billion, and completion of a successful study within a fundable budget was judged not to be feasible.

In contrast, more than 15 years after their initiation, the Danish National Birth Cohort and Norwegian Mother and Child Cohort Study are well on the way to providing important evidence on the conditions they set out to investigate. More than 800 articles have been published from data emerging from the 2 cohorts. Key findings include studies on the importance of maternal chronic diseases in pregnancy⁵ to child health and evidence that folic acid intake by the mother periconceptionally might be related to occurrence of autism spectrum disorder.⁶

For some diseases, in particular cancer, type 1 diabetes, and cerebral palsy, it was apparent at the planning stage of these cohorts that even 100 000 participants would be marginal for providing the required power. Consequently, the Danish National Birth Cohort and Norwegian Mother and Child Cohort Study have participated in an initiative to pool data with some older studies that have relevant data to obtain the necessary power. These include the Jerusalem Perinatal Study, the

Corresponding

Author: Terence Dwyer, MPH, MD, MB, BS, AO, The George Institute for Global Health, Oxford Martin School, University of Oxford, 34 Broad St, Oxford OX1 3BD, United Kingdom (terence.dwyer@georgeinstitute.ox.ac.uk).