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Food as Exposure

Nutritional Epigenetics and the Molecular Politics of Eating

FROM FRUITS ENGINEERED TO BE HIGH IN anthocyanins to cheese sticks fortified with omega-3 fatty acids, supermarket shelves are increasingly stocked with foods bearing a health message about their constituent molecules. Even if you don't know an antioxidant from a trans-fat, you're likely to be able to categorize these two molecules as good or evil. Ever since the first words about cholesterol were allowed to appear on packages of rolled oats, the peculiar rhetoric of the health effect that makes no specific claims has been under intense development, and

it has developed into a fine art. Since 1997 nutritional supplements have been allowed to bear general claims about "supporting immune function" or "strengthening the body's defenses" as long as they include this text: "This statement has not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease." Conventional foods as well as nutritional supplements are allowed to make "qualified" health claims if they can "establish a relationship between a food or food component and a disease or

health-related condition," such as high blood pressure.

In general, this linking of particular food molecules to health is part of a peculiarly twenty-first century synthesis of food and medicine. Food has been understood to be medicinal in many different ways in different historical periods; one has only to consult the Roman physician Galen on the qualities of lentils to see that it is not new to think about food as more than fuel for the body. According to *The Properties of Foodstuffs*, twice-boiled lentil soup will dry up gastric

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fluxes and increase the tone of the esophagus, and is thus an appropriate food for those with gastric complaints. However, in our time, an intensely *molecular* language of interaction between food and the body is in play. It is the interaction of food's molecules with the body's molecules that draw the focus of biomedical research, and, in consequence, shapes regulatory cultures of food as well

as the marketing language and popular perception of food's therapeutic or disease-preventing effects.

As a social scientist, I am interested in the increasingly molecular focus of contemporary eating discourse, and the attendant anxieties and uncertainties produced by seeing food as both potential preventative medicine and potential threat to health. What are

the political and social consequences of the sciences of food and metabolism used to establish a relationship between food and health? My studies are directed at some of the places in which the language and logic of food as molecular medicine is currently under construction, and I have focused my attention on the biomedical research laboratory. I have been looking at nutritional epigenetics, an



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area of biomedical research that seeks to link nutrition with patterns of gene expression. The basic hypothesis of nutritional epigenetics is that food type and availability during so-called “critical periods” of development affect patterns of gene expression and thus the physiology and disease susceptibility of an organism for the rest of its life, and perhaps the life of future generations. Thus,

the findings produced by this research are particularly significant in relation to prenatal and early postnatal nutrition. As such, the politics of parenting are inextricably entwined with even the most esoteric-seeming experiments in the molecular biology of metabolism.

Nutritional epigenetics seeks to understand how the molecules in food affect long-term

health through their interaction with the molecules that mediate gene expression in the body. Much of the work done in this area is done in animal models. In particular, the agouti mouse model has been used to establish a link between prenatal and early postnatal nutrition and gene expression. The agouti mouse model has a piece of foreign DNA called a retrotransposon in the promoter region of the agouti gene, which normally produces the agouti protein in select cells in the body and affects coat color. If the retrotransposon is heavily methylated—has many CH₃ groups attached to the cytosine molecules in the DNA sequence (the C's in ATCG)—then the gene is not expressed at high levels. However, if the retrotransposon is not methylated, the DNA is open to other molecules that spur the transcription and translation of the gene into its protein product, and the agouti protein is expressed all over the mouse body. This wide expression of agouti protein has several effects on the mouse's body—most noticeably it is fat and yellow instead of thin and brown, and it suffers from susceptibility to diabetes and cancer. Significantly, two mice can be genetically

identical, but have very different bodies, because the set of molecules attached to the DNA are different and thus modulate the same genes differently. These changes are referred to as “epi”-genetic, exactly because they do not affect the gene sequence in the same way as a mutation would, but work instead at the level of regulation.

Pregnant agouti mice fed diets supplemented with substances that contribute methyl groups to the methylation process produce litters in which a higher proportion of the pups have brown, thin bodies than their non-supplemented peers. Pups weaned and then fed a diet depleted in methyl donors show loss of methylation of genes associated with diabetes. Even if the mice are switched back to a methyl-donor sufficient diet after 60 days, the effect on gene expression lasts through their lifetime (Waterland et al. 2006). These molecular effects of diet have also been found in human populations—individuals who were in utero during the Dutch hunger winter of 1944-1945 show reduced methylation of the same gene locus associated with diabetes in comparison with their siblings born after the war (Heijmans et al, 2008). The basic

idea is that food conditions early in life, in utero or early postnatal life, affect patterns of gene expression and thus the way the body works for a lifetime, and perhaps beyond. There is even some evidence that methylation patterns are heritable between generations of mice. Scientists call these animals “epigenetic biosensors,” because their bodies show at a macroscopic scale events happening at a molecular scale.

These scientific developments mark a pronounced shift in our understanding of both food and metabolism. Where classic biochemical studies of metabolism depicted food as fuel or as substrate for the building blocks of the body, nutritional epigenetics depicts food as information or molecular signal. This information, conveyed by the molecules traveling from the outside of the body to the inside, indicates what kind of environment the body will grow up to occupy and thus affects the systems that respond to the environment. In the logic of nutritional epigenetics, food at critical periods in development actually shapes the metabolic interface that will process food later in the life of the organism. Or, to put it most



By looking at how something social, such as “eating,” is conceptualized and constructed as an experimental variable, we can track the ways in which the social is given scientific materiality, and the ways, in turn, material scientific objects are then taken as meaningful for social life and can change understandings of such broad concepts as “environment” or “food.”

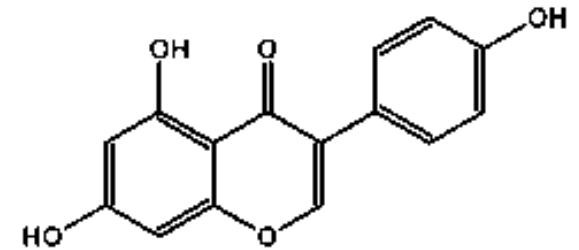
bluntly, food shapes the conditions of its own reception in the future. Food, as a carrier of biologically significant molecules, is a kind of environmental exposure that shapes the way the body responds to the environment.

When scientists do experiments, they are creating highly controlled situations to test particular hypotheses about the effects of food on health. By looking at how something social, such as “eating,” is conceptualized and constructed as an experimental variable, we can track the ways in which the social is given scientific materiality, and the ways, in turn, material scientific objects are then taken as meaningful for social life and can change understandings of such broad concepts as “environment” or “food.” When experiments are done with animal models such as the agouti mouse, the controlled setting that is created in the laboratory is what I call an “experimental image” of human life, and in this experimental image an emerging discourse of food as exposure becomes very clear.

In the agouti mouse model system, three substances in particular characterize the experimental image: soy, folic acid, and bisphenol-A. Soy-derived genistein is one of

the molecules fed to these mice. Agouti mice whose diets were supplemented with genistein had offspring whose coat colors shifted toward brown, indicating increased methylation of the agouti promotor (Dolinoy *et al.* 2006). Genistein is a socially significant molecule, because soy is everywhere. Soy is consumed in the form of soy milk and tofu and edamame; it is a commonly used ingredient to increase protein content or lend texture and form to foods; it is fed to industrially farmed chickens and pigs. It is also frequently used as a base for infant formula. Of course, the experiment does nothing more than signal that soy in the diet can affect gene methylation; it does not indicate whether this is a good, bad or neutral occurrence, nor what kinds of dosage might have health effects, nor whether these effects happen in humans in the same way as mice. Nonetheless, the results seem highly applicable to human affairs exactly because of the ubiquity of soy.

A second socially significant molecule that feeds these experimental animals is folic acid, which provides methyl groups to the metabolic cycles that methylate DNA. Folic acid is probably a familiar micronutrient for



Top, soybeans; below, molecular structure of genistein.

readers. Because folic acid supplementation has been shown to decrease the incidence of neural tube defects and anencephaly when consumed in the first trimester of pregnancy, mandatory folic acid fortification of all wheat products in the United States was instituted as a public health measure in 1998. Currently over 65 countries world-wide have mandatory fortification of wheat or maize flour or both.

Folic acid is a synthetic form of a molecule whose natural form is called folate. Folate is in high levels in things like oranges, beans, and leafy green vegetables. Folic acid is in all wheat products, and is added to many “functional foods” such as nutrition bars. Consumers have little control over the amount of folic acid they consume, as it is not a mandatory part of food labeling to indicate how much is in a serving of a bread or cereal product—only the original fortification is mandated. Pregnant or periconceptual women, meanwhile, are encouraged to take an additional folic acid supplement of 100% RDA on top of their regular diet and their regular intake of fortified foodstuffs. Now, more than ten years after mandatory

fortification came into place, unmetabolized folic acid is present in the blood of most individuals—particularly children and the elderly who consume higher proportions of cereal and bread (Smith et al. 2008). The rate of neural tube birth defects has dropped markedly in the United States since the introduction of this policy. Debate has begun to reopen however, as questions are raised about links between methylation and cancer, and excess folic acid could be responsible for causing or exacerbating colon cancer in older adults, even as it prevents birth defects.

The idea of food as exposure is intensified by the slippage between the notions of food and toxin. Also using the agouti model, researchers fed mice high levels of bisphenol-A, observed the depression of methylation caused by it, and then attempted to counter the effect with methyl-supplementation (Dolinoy 2007). Bisphenol-A is a plastic-derived endocrine-disrupting molecule that is ingested through food and drink, because it is in the linings of cans and plastic containers used for storage and transport. Food can therefore be biologically

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active in terms of epigenetic changes in its “natural” state, in its manufactured or engineered state, and in its unintentionally polluted form.

Genistein and folic acid are pervasive and invisible ingredients rather than discrete and obvious components of food, and bisphenol-A is also undetectable and widespread. There are clear implications for conceptualizing food as a discrete object that can be refused versus food as a miasma in which people are immersed. The first frame—food as controlled by individual choice—implies that personalized nutrition will be part of personalized medicine, drives the production of consumables for health, and increases the imperative to monitor food intake at the molecular level, thus increasing the susceptibility of publics to molecularized marketing. This is the frame in which many functional foods operate: consumers are encouraged to take care of themselves by buying and eating foods with tangible health benefits, such as the promise that one pot of yoghurt a day equals one person’s improved digestion/immune function. The second frame—food as milieu—leans instead toward

a sense of collective or social responsibility for health and regulatory approaches, as these are things well beyond our control as individuals. If people are encased within these environments, then all one can do is work on those environments and the amelioration of their effects and do it in a way which affects more than one individual at a time.

Nutritional epigenetics, in linking food and food pollutants to health, has the potential to contribute to both an individualized politics of eating and an increased sense of social responsibility for caring for the food environments of populations. It might seem yet another way to pin responsibility for fetal and child health on the individual actions of pregnant women, but the politics of this particular linkage of eating and long-term health points in a rather different direction. The logic of epigenetics spreads the responsibility over many generations as well as between men and women, as epidemiological studies point to an important role of male nutrition in influencing epigenetic regulation in offspring (Kaati et al, 2002). Analysis of the experimental images being generated in the laboratory indicates that the kinds of objects

that are chosen to represent “food” in these controlled settings contribute strongly to a notion of food as a kind of molecular cloud that surrounds us. However, this science is relatively new and so far its main impact has been to shift the way food is studied in molecular biology and epidemiology rather than to shift the way food is controlled or perceived in spheres of regulation or consumption.

Epigenetics raises questions about the governance of food and the environment more generally in the interests of human health. It remains to be seen how or whether this will translate into regulatory changes, for example, banning the use of Bisphenol-A in food containers, a rethinking of the regulation of supplements and functional foods, or renewed attention to the public health impact of improving prenatal care. Or how it might translate into individual action: as epigenetics becomes more familiar to public audiences through newspaper accounts, food health claims, and popularizations, it will be important to track how consumers incorporate this narrative into their food choices and their understandings of food and

the body. At its heart, nutritional epigenetics represents a hope for intervention in the long-term health of bodies and thus the general health of populations, via the medium of food. Epigenetics has a very specific temporal logic to it, one that emphasizes the multigenerational impact of the environments surrounding fetuses and children. Given the social and cultural importance of food and eating and the fraught nature of contemporary parenting when it comes to feeding children, the unfolding generation of these specific links between nutrition and health calls for our continued critical attention.

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