ELSEVIER

Social Science & Medicine 65 (2007) 1730–1741

SOCIAL SCIENCE & MEDICINE

www.elsevier.com/locate/socscimed

How much can a large population study on genes, environments, their interactions and common diseases contribute to the health of the American people?

Claudia Chaufan

University of California Santa Cruz, Santa Cruz, CA, USA

Available online 5 July 2007

Abstract

I offer a critical perspective on a large-scale population study on gene–environment interactions and common diseases proposed by the US Secretary of Health and Human Services' Advisory Committee on Genetics, Health, and Society (SACGHS). I argue that for scientific and policy reasons this and similar studies have little to add to current knowledge about how to prevent, treat, or decrease inequalities in common diseases, all of which are major claims of the proposal. I use diabetes as an exemplar of the diseases that the study purports to illuminate. I conclude that the question is not whether the study will meet expectations or whether the current emphasis on a genetic paradigm is real or imagined, desirable or not. Rather, the question is why, given the flaws of the science underwriting the study, its assumptions remain unchallenged.

Future research should investigate the reasons for this immunity from criticism and for the popularity of this and similar projects among laypersons as well as among intellectuals.

Keywords: Type 2 diabetes; Social inequalities in health; Genetics; Reaction norm; Geneticization; USA

Introduction

> Genetics is playing an increasingly important role in the diagnosis, monitoring, and treatment of diseases [...]. The potential for using genes themselves to treat disease—gene therapy—is the most exciting application of DNA science.
>
> The Human Genome Project Information (http://www.ornl.gov/sci/techresources/Human_Genome/medicine/medicine.shtml)

In May 2006, a Task Force on Large Population Studies called upon by the US Secretary of Health and Human Services' Advisory Committee on Genetics, Health, and Society (SACGHS) issued a report with the intention of evaluating the feasibility of a project to study "genes, environments, their interactions and common diseases" (Secretary's Advisory Committee on Genetics, 2006). The report claimed that "characterizing human genetic variation and how genetic variants interact with environmental factors (physical, behavioral, and social) to influence health is currently *one of the most pressing goals* for scientists trying to unravel and understand the underlying causes of common diseases", and

E-mail address: claudiachaufan@yahoo.com

0277-9536/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.socscimed.2007.05.049

that "scientists hope that *major clinical and public health advances will be realized by learning where variation among individuals lies within the genome, how it differs among healthy, predisposed, and sick individuals, and how particular variants of DNA interact with each other and diverse environmental factors*" (emphasis added, p. 4). The challenges of the project notwithstanding—involving a large number of research subjects, and consuming a considerable amount of the budget of the sponsoring agencies, the US Department of Health and Human Services (US DHHS) and the National Institutes of Health (NIH)—the SACGHS believed it had "the potential to generate significant health benefits" (Secretary's Advisory Committee on Genetics, 2006, p. 10).

In June 2006, I received an invitation to comment on the project, yet what started as my reply developed into an analysis of two major claims of the proposal, namely, that examining gene–environment interactions is a pressing goal for scientists concerned with human health, and that this examination will generate significant health benefits. I divide this analysis into four parts: first, I discuss the geneticization debate, locating it within a broader nature–nurture debate, and argue that geneticization scholars have missed the target of their critiques, by failing to understand how the strengths and limits of genetic knowledge affect its potential contributions to human health; second, I examine the relevance of the proposal to primary, secondary, and tertiary prevention; third, I clarify key concepts in genetics; and fourth, I discuss the implications of the project for public health policy, and the resilience of what appears to be its underlying disease paradigm to be challenged on its own terms, even by critics.

This type of analysis, I believe, is lacking in both the medical and the sociological literatures, and in this paper I try to address this gap. I use type 2 diabetes (henceforth diabetes) as a paradigmatic "common disease" of the sort that the proposed project attempts to illuminate, yet I expect this analysis to be relevant to conditions comparable to diabetes in their pathophysiology, the environments promoting them, and the risks they pose to human health, such as common, multifactorial forms of cancer, hypertension, or heart disease. I imply all these when I refer to "common diseases".

Diabetes today is common indeed, and there are many good reasons to be concerned about it. First, if left untreated or if poorly treated, diabetes leads to disabling complications and to premature death. Second, rates of diabetes have increased epidemically over the last ten years, the increase is global, and its projected distribution is very uneven. By the year 2025, the number of people affected by diabetes will have risen to 300 million (from 135 million in 1995), and while the increase will be of 42% in the developed countries, it will reach 170% in developing countries (King, Aubert, & Herman, 1998). While these numbers do not discriminate among types of diabetes, at least 90% of the cases are presumed to be type 2, the real protagonist of the epidemic. Third, diabetes is very costly: in the United States alone, it imposes a toll of over 130 billion dollars—one out of every ten health-care dollars (American Diabetes Association, 2003). Last, rates of diabetes and diabetes complications are two to six times higher among minorities worldwide than among dominant groups (American Diabetes Association, 2001). Clearly, anything that contributed to fighting diabetes would be worth considering. And yet, I believe that a study of the sort proposed will achieve nothing to this effect—it would be redundant at best, while at worst it would distract from the real roots of, and solutions to, common diseases.

The Nature–Nurture debate: a reenactment

The debate about which "part" of the range of anatomical, physiological, or behavioral features of human beings is caused by nature and which by nurture—or by some interaction of the two—is certainly not new. While for Plato, whether individuals were meant to be artisans, soldiers, or philosopher-kings, depended on their "essences" being bronze, silver, or gold (Bloom, 1968), for 19th century European aristocrats it was a matter of "blood". Thus readers of Oliver Twist "knew" that the protagonist's perfect English and manners, much like the defective English and manners of the Artful Dodger, were the result of nothing but biological pedigree (Lewontin, Rose, & Kamin, 1984). One incarnation of the "nature–nurture" debate, more suited to the modern world than "essences" or "blood", is the "genes–lifestyle" debate, premised on the idea that genes contribute, at least in part, to conditions ranging from diabetes, to heart disease, to cancer, and that identifying this contribution is crucial to fighting these conditions (Chaufan, 2006).