

THE ELEPHANT IN THE ROOM: THE INVISIBILITY OF POVERTY IN RESEARCH ON TYPE 2 DIABETES

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ABSTRACT

Over two hundred years of anecdotal, epidemiological, and experimental evidence indicate that poverty breeds disease. This holds true for type 2 diabetes, which both in the United States and other developed nations disproportionately occurs, cripples, and kills among the poor. In this article we examine rhetorical strategies used in 30 journal articles indexed under type 2 diabetes and poverty. As we show, poverty is rarely highlighted in this literature as a causal factor. Instead, explanations for diabetes among poor people overwhelmingly emphasize features of patients—their biology, behaviors, psychology, culture, or other “risk factors”—while ignoring, reframing or neglecting the links between poverty and disease. By so doing, these discursive strategies naturalize higher rates of diabetes among poor persons, legitimize relations of domination in the larger society, and encourage only research projects, treatment practices and health and social policies that do not challenge existing social relations. We discuss the implications of these discursive practices for medical research and care, and for social and public health policies.

PERSONAL REFLEXIVE STATEMENTS

Claudia Chaufan

In the late nineties, as a newcomer to America trained as a clinical diabetologist in Argentina, a job as a diabetes educator for Latinos opened my eyes to a political reading of diabetes and of diabetes inequalities. As I learned about Latinos’ life and health chances and contrasted them against public discourses around their “high risk” status—built around ill-defined “genetic predispositions” and decontextualized “lifestyle choices”—I opted for an

academic career in sociology, became a health care activist, and never looked back. Human health is the product of countless interactions between individuals and the historical sequence of environments—physical, social, political—they encounter over the life course. Type 2 diabetes is a particularly sensitive indicator of this process, and so a biologically informed and socio-politically grounded account of this disease can serve as a model to examine, understand, and act more effectively and justly on comparable conditions affecting overwhelmingly socially excluded groups. This is my goal as an intellectual, teacher, and activist: to contribute to the greater visibility of the fundamental social and political causes of health inequalities, to highlight the implications of these inequalities for human rights, and to inform more effective and just public policies.

Rose Weitz

*My interest in the impact of social inequality on health began at the age of 17, when I first had to negotiate the New York City public hospital system with my highly asthmatic new husband. The chaotic scenes, long waits for people in obvious pain, and abrupt treatment given to most patients, coupled with the obviously more respectful treatment given to my husband and I (who were often the only whites in the waiting room), proved an eye-opening experience for someone like myself who was raised with middle-class expectations of health care. I was a college freshman at the time, and soon began taking courses in sociology, including sociology of health. The emphasis on power dynamics within health and health care made sense to me immediately, and I have spent my career investigating these issues with regard to topics as varied as home birth in the United States (in *Labor Pains: Modern Midwives and Home Birth*), the impact of stigma on the lives of people with AIDS (in *Life With AIDS*), and the invisibility of poverty in medical research on diabetes.*

Over two hundred years of anecdotal, epidemiological, and experimental evidence indicate that poverty breeds disease, even as the major types of diseases and the particular mechanisms linking poverty with disease have changed (Link and Phelan 1995; Raphael 2007). Early work in the tradition of social medicine led researchers and practitioners such as Louis Villerme and Rudolf Virchow to suggest that the best way to reduce disease was to provide poor persons with better living conditions and greater political power (Waitzkin 1981). As we show, this understanding of disease "causes" and "treatment" has been lost in current research on diabetes and poverty.

In this article, we look at how research articles published in health-related journals—the "public face" of the health research community—explain the

causes of diabetes, pre-diabetes, and diabetic complications among poor persons. We find that poverty is rarely highlighted in this literature as a causal factor. Instead, explanations for diabetes and its complications overwhelmingly emphasize features of patients (their biology, behaviors, psychological states, or culture), focus on identifying “risk factors,” and recommend “targeting” and changing individuals, while failing to challenge risk-producing environments and reframing, neglecting or ignoring the links between poverty and disease.

By so doing, these explanations turn poverty into a proverbial “elephant in the room,” whose existence everybody recognizes but ignores so they can continue with “business as usual.” These discursive strategies, by concealing the impact of poverty, naturalize higher rates of diabetes among poor persons and legitimize relations of domination in the larger society. We conclude with a discussion of how framing diabetes inequalities as a “problem” of poor diabetics hinders or misdirects health care research, practice, and policy as well as broader social and economic policy.

BACKGROUND

Since WWII, rates of diabetes have skyrocketed in the United States, as elsewhere (<http://www.diabetes.org/diabetes-statistics.jsp>). Type 2 diabetes, the focus of this paper, accounts for about 90 percent of all cases of diabetes, and affects an estimated 19 million Americans (seven percent of the population). An additional 54 million Americans suffer from pre-diabetes—abnormal blood glucose levels high enough to impair health irreversibly, but not high enough to meet the current diagnostic criteria for diabetes. In total, one-quarter of the U.S. population has either pre-diabetes or type 2 diabetes (henceforth referred to jointly as *diabetes*).

The increase in rates of diabetes has brought tremendous costs for individuals and for society: in 2002, individual medical expenditures among Americans with diabetes (type 1 or type 2) were more than twice those of comparable non-diabetic Americans, and diabetes accounted for almost one-fifth of all U.S. health care expenditures (American Diabetes Association 2003). Additionally, diabetes is associated with premature death and crippling disability, including blindness, kidney failure and foot amputations. All this has led experts to suggest that diabetes is “the epidemic of the New Millennium” (Jovanovic 1999: 33).

The explosion of type 2 diabetes has taken a toll on all social groups, affecting “the old and the young, men and women, all racial and ethnic groups, the rich and the poor” (Diabetes Research Working Group 1999:15). Nevertheless, both in the United States and in other developed nations, diabetes has taken a disproportionate toll on minorities and on the poor. For instance, among African Americans, Hispanics, Native Americans and Pacific Islanders, rates of diabetes

and of its complications (including blindness, kidney failure, and heart disease) are two to six times greater than among white Americans (Carter, Pugh and Monterrosa 1996). Among poor Appalachians, diabetes is increasing at a disturbing speed among whites and ethnic minorities alike (Wright 2003). Similarly, a major Japanese study found that eight times as many low-status workers as high status workers had diabetes (Morikawa et al. 1997), and a Finnish study found that, among diabetic males, almost twice as many blue-collar workers as white-collar workers died in a five-year period (Forssas et al. 2003).

There is virtual agreement in the medical community that across ethnic and socioeconomic groups the rise in diabetes results from greater access to calories coupled with the physically undemanding nature of modern-day life, which combine to cause insulin-resistance, the basic cellular malfunction underlying diabetes. Similarly, there is near-unanimous agreement that appropriate diets and physical activity dramatically reduce risk of diabetes, irrespective of ethnicity (Tuomilehto and Lindstrom 2003).

But why are poor and minority populations so much more vulnerable to these processes than others? While the mainstream medical literature typically describes diabetes as “multifactorial” (McCarthy and Hattersley 2001; Hansen 2002), suggesting that among the many factors, it is “affluence” that drives the current epidemic (Fall 2001), a smaller but well-documented set of writings suggest that it is *poverty* that directly causes not only the chronic complications resulting from poorly controlled diabetes, but diabetes itself (Silverman et al. 1995; Benyshek, Martin and Johnston 2001; Ben-Shlomo and Kuh 2002; Branca and Ferrari 2002; Moore 2002; Aerts and Van Assche 2003; Barker 2003; Benyshek et al. 2004). In practice, however, most popular and medical explanations for diabetes pay little if any attention to how poverty as a systemic factor causes diabetes, and instead focus on the biology, psychology, or culture of affected individuals—so-called risk factors (Chaufan 2006).

NURTURE BEGINS AT CONCEPTION: THE DEVELOPMENTAL ORIGINS OF TYPE 2 DIABETES

That type 2 diabetes is rooted in biology is undeniable: the disease develops when a malfunctioning pancreas, the producer of insulin, is faced with insulin resistant tissues and so no longer can maintain an individual’s blood glucose levels within a healthy range. Left unchecked, chronically elevated glucose ultimately can lead to serious illness or disability, including blindness, kidney failure, foot amputations, and nervous system disease, as well as to early death (<http://www.diabetes.org/diabetes-statistics/complications.jsp>).

But what causes insulin resistance in the first place, and why are some populations so much more at risk than others? Reflecting the widespread adoption of a genetic paradigm to explain diseases in general over the last quarter century (Conrad 1997), the most common biological explanation for diabetes in both popular and medical discourse is that a “genetic predisposition” leaves certain populations more at risk for diabetes than others when exposed to both sedentary lifestyles and high calorie diets (Saudek, Rubin and Shump 1997; Bernstein 2000; American Diabetes Association 2001; Marks 2004; Caballero 2006). In addition, cultural explanations for the disproportionate rates of diabetes focus on long-standing traditions and beliefs within minority cultures allegedly leading to unhealthy behaviors. For example, some have argued that Latino cultures equate chubbiness with health and keep individuals from adopting lower calorie diets (Blixen, Singh and Thacker 2006; Caballero 2006). Psychological or behavioral explanations for the disproportionate burden of diabetes in poor and minority populations often use similar variables, but tie them to intrapsychic, rather than cultural, processes, arguing, for example, that low self-efficacy keeps poor individuals from committing to healthier diets (Glasgow, Toobert and Gilletteet 2001).

Not surprisingly, explanations centered on individuals lead to interventions targeting individuals, even if in large numbers. For example, one national campaign sponsored by the US Department of Health and Human Services and geared to reduce ethnic disparities in health “encourages the public to support loved ones living with diabetes by working with them to practice a healthy lifestyle and [to] get regular checkups” (US Department of Health and Human Services and National Institutes of Health 2005). Another national campaign, “The Power to Control Diabetes Is in Your Hands,” aims at increasing “public awareness” and “empowering older adults” with its brochure, the “Power to Control,” which stresses the “importance of keeping blood glucose close to normal levels” as a strategy to help “older adults with diabetes [...] on fixed incomes” (National Diabetes Education Program, National Institutes of Health 2005).

The non-profit sector itself often concedes to this individualistic discourse. The American Diabetes Association, the largest non-profit in America concerned with “preventing and curing diabetes, and improving the lives of all people affected by diabetes,” offers readers of *Diabetes Forecast* “8 coverage tips” to deal with un-insurance (Diabetes Forecast, March 2006). From inside the covers of the Spanish edition of the magazine, Latino dietitian Gloria Rodrigues Mulloy notes the “alarming trend toward obesity” among Latinos and their children, asserts that the “most effective [nutrition] education” begins at home, and advises parents to “plan ahead,” to “make sure [their] pantry isn’t a source of

temptation,” and to patiently insist that their children eat their vegetables (Cummings 2005: 21-25).

Socioeconomic explanations for the high rates of diabetes found among poor persons, whether minorities or non-minorities, are considerably less common. Yet a substantial body of medical research describes how poverty can directly cause insulin-resistant states, pre-diabetes, and, eventually, diabetes (Silverman et al. 1995; Benyshek et al. 2001; Ben-Shlomo and Kuh 2002; Branca and Ferrari 2002; Moore 2002; Barker 2003). When a pregnant woman is malnourished, her fetus also receives inadequate nourishment. This affects the development of the fetal pancreas leading to largely irreversible glucose intolerance, i.e., an in-born (but not genetic) biological predisposition to diabetes. Recent animal experiments offer evidence for this process, even after controlling for genotype (Benyshek 2006).

Similarly, very young children, whose pancreatic function is still developing, are especially vulnerable to becoming insulin resistant if they are poorly nourished. If that baby or young child is female and eventually becomes pregnant, the resulting fetus will be exposed to its mother’s insulin-resistant state. Like the previous cases, this exposure can impair the fetus’s pancreatic development and lead to diabetes later in life (Silverman et al. 1995). This cycle can continue over generations, reproducing a biological predisposition to diabetes that can be triggered or compounded by multiple pregnancies, high calorie diets and low levels of physical activity (Ben-Shlomo and Kuh 2002; Branca and Ferrari 2002; Aerts and Van Assche 2003; Barker 2003; Benyshek et al. 2004). In the developed world, all these factors are most common among the poor. Taken together, these forces lead to the intergenerational, biological (but not genetic), transmission of diabetes, and may explain the staggering rates of diabetes among southwestern Native Americans (Benyshek et al. 2001) as well as the explosion of diabetes in India and China (Moore 2002), where many individuals born to chronically malnourished rural women have migrated to cities and adopted, whether willingly or for lack of better options, a sedentary, high-calorie lifestyle.

At the same time, regardless of whether individuals have been “wired in the womb” for diabetes, they have the best chance of escaping its worst complications if they have access to healthy foods, opportunities for exercise, necessary medication, technologies for delivering those medications effectively, and health care for the complications that can develop even with full medical compliance. Far fewer poor persons with diabetes have access to these resources, leading to much higher rates of complications, disability, and death among these individuals than among more affluent persons with diabetes. Indeed, the experience of deprivation itself, over the life course—especially

common among Aboriginal populations and the excluded—can affect sugar uptake and inhibit the effects of insulin (Marmot and Brunner 2001). This occurs through both central physiological mechanisms and behavioural coping mechanisms.

Yet notably, even when the role of poverty in the causation of diabetes or its complications is publicly recognized, poverty itself is typically framed as a “problem” of poor people, who require “special interventions.” For instance, in a study that stratifies patients according to their risk of developing diabetes complications Nicolucci et al. conclude “in the definition of risk profile for each individual patient, socioeconomic status [...] needs to be taken under serious consideration, since [it] can determine a complication risk not dissimilar from hard clinical variables, such as hypertension and diabetes duration” (Nicolucci, Carinci and Ciampi 1998:1439). The authors recommend designing and implementing “specific educational interventions *targeted* [emphasis added] to the socially disadvantaged strata of the population” (Nicolucci et al. 1998: 1439).

DATA AND METHODS

Sample

Because our goal was to understand how diabetes and its relationship to poverty was presented to and by health care practitioners and researchers, we began by searching for articles jointly indexed in Pub Med under both “poverty” and “diabetes mellitus type 2” as major subject headings.¹ (Major subject headings are those identified by authors of journal articles or by the National Library of Medicine’s expert librarians as *primary* topics in an article.) We chose “poverty” as our search term because the National Library of Medicine defines it as “a situation in which the level of living of an individual, family, or group is below the standard of the community,” which was precisely what we were interested in investigating. “Poverty” was introduced as a subject heading in 1968.

To identify appropriate articles, we first searched PubMed for all research articles (i.e., not news reports, letters, or editorials) written in English. This initial search produced too few articles for a workable dataset. Consequently, we broadened our search to articles written in English that listed “diabetes mellitus, type 2” as a *major* subject heading and poverty as a *non-major* subject (MAJR versus MeSH, in PubMed’s terms). This search, completed in November of 2006, produced 55 articles. We further refined our search by eliminating one news report that had eluded PubMed’s filters, and eight articles that addressed the *consequences* of diabetes or its complications (such as depression, poor body image, or high emergency room utilization) rather than the *causes*. Finally, we

eliminated 16 articles that reported on research from outside the United States because elsewhere the meanings and consequences of such basic concepts as poverty and limited access to health care are different enough to invalidate a comparison.²

As the definition of our sample suggests, we included all PubMed-indexed articles that met our sample criteria regardless of their field of publication. Fifteen were published in medical journals, 10 in nursing or diabetes education journals, 3 in public health journals and 2 in social science journals. We based this decision on the particular nature of diabetes care, in which at least theoretically a team approach is promoted. And because overwhelmingly diabetes care is self-care, the separation between medical management, nursing care, and patient education is vague at best. Additionally, the first author's experience in medical practice and in diabetes education suggests that to an unusual extent practitioners across fields read each other's journals. This left us with a final sample of 30 articles.³

Data collection and analysis

To analyze our sampled articles, we developed a code-sheet of 18 closed-ended questions designed to elicit for each article the authors' views on 1) the causes of diabetes, pre-diabetes, or diabetes complications, 2) the best ways to attack these problems, and 3) the nature of the link between poverty and these problems. These issues were central in some articles but tangential in others, yet all articles at a minimum included introductory or concluding remarks that put their research in broader context and provided an overview assessment of causation and potential solutions. The initial version of our code-sheet was based on our background reading and knowledge of the relevant social science literature and on the sociological and clinical experience with diabetes of one of the authors.

We "field-tested" this code-sheet with five initial articles, adding and fine-tuning questions and possible answers as needed to fully capture the range of concepts included in these articles. We repeated this process with the next five articles, in order to reach agreement on the nature of our questions and categories, but not on our coding for the articles themselves. Once the code-sheet was finalized, each of us individually coded each article, achieving a concordance rate of 87%, considered reasonable for this type of analysis (Lombard, Snyder-Duch and Bracken 2002). We subsequently discussed the differences in our coding, further clarified the meaning of our code-sheet questions and answers and reached agreement on all codes.⁴

FINDINGS

Our first finding was how few articles were indexed in PubMed under both poverty and diabetes. Between 1968, when PubMed first began indexing articles under the term *poverty*, and late 2006, when we conducted our search, slightly over 30,000 articles had been indexed using diabetes type 2 as a major subject heading. Yet only 21 of these used poverty as a major heading as well. This in itself illustrates how little attention has been devoted to the link between diabetes and poverty.

This finding was replicated in our expanded and refined sample (using poverty as non-major subject heading) of 30 articles. While all were identified by their keywords as focusing on both poverty and diabetes, poverty as a cause of diabetes or its complications seemed near invisible within their pages. The articles typically began with a brief discussion of the epidemiology of diabetes and mentioned that diabetes and poverty were correlated, but made no mention of any causal links. For example, one article stated that “non-insulin dependent diabetes mellitus...is becoming alarmingly common among certain sectors of the population, particularly the ethnic and minority poor” (Hunt et al. 1998: 656), and another that the “prevalence of the disease and its physical, psychological and economic impacts...appear greatest for the poor” (Hopper and Schechtman 1985: 275). Use of passive voice further obscured causation, as in statements such as “The highest prevalence of diabetes has been observed in women, older people, the less educated, and low-income families” (Rimmer et al. 2002: 571).

Of the 30 articles we reviewed, all of which focused on diabetes among the U.S. poor, only 12 (40%) described poverty as explicitly contributing to diabetes or its complications. For example, Rhee et al. reported that among poor African Americans, “The most common reason for having trouble obtaining medical care was insufficient funds, cited by 78% of patients” (Rhee et al. 2005: 738). Even among these 12 articles, only three (10% of the sample) described poverty as the *primary* cause of diabetes (Tables 1 and 2). Another 3 articles described insufficient access to medical care as the primary cause of diabetes and its complications but did not identify poverty as the cause of this insufficient access, and 16 described lack of access to health care as either a contributing or a primary factor in diabetes or its complications.

How, then, did the other articles, all indexed under poverty, explain why diabetes and its complications are disproportionately common in poor populations? Most commonly, the explanations focused on poor individual choices. We coded articles as falling in this category if they primarily explained these health problems as resulting from problematic individual choices *and* linked those choices to ignorance, psychological characteristics, or idiosyncratic cultures *rather than* to poverty or lack of health care. For example, one article

Table 1: Primary Cause of Diabetes, as Identified in 30 PubMed-Indexed Articles on Diabetes among the Poor

Primary cause	N	%
Poor individual choices (independent of culture or poverty)	13	43%
Minority culture (not explained by poverty)	6	20
Insufficient health care access (not linked to poverty)	3	10
Poverty	3	10
Genetics	0	0
Other	0	0
Multiple or unclear answers	5	17
TOTAL	30	

Table 2: Factors Contributing to Diabetes, as Identified In 30 PubMed-Indexed Articles on Diabetes among the Poor*

Factors	N	%
Ethnicity (not linked to poverty)**	10	33
Insufficient health care access (not linked to poverty)	16	53
Poverty	12	40
Health care provider attitudes or behaviors	11	37
Genetics (linked to ethnicity or not)	6	20

*Answers are not mutually exclusive

** Includes minority culture, minority genetics, and minority low-knowledge levels

focused on how mistrust of health care workers leads patients to ignore recommendations regarding diet and medication (Egede and Michel 2006). The article did not address other possible explanations for medical noncompliance. Nor did it address whether “medical mistrust” might have resulted from the care these poor patients received from health care workers or from their realization that they could not control their diabetes despite complying to the best of their abilities. Instead, medical mistrust was described purely as a psychological attribute.

Similarly, most articles linked diabetes and its complications to problematic choices made independently from patients’ poverty. This tendency was reinforced by the ways authors interpreted their data. Twenty-one of the 30 articles reviewed included reports from subjects on barriers that made it difficult for them to comply with medical advice about diabetes. However, four of these 21 articles (19%) reported their data in ways that dismissed subjects’ belief that poverty posed a real, rather than a perceived, barrier to medical adherence.

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For instance, one article on poor African Americans who have diabetes reported that “Subjects *felt* that they could not afford to buy low-sugar or low-fat foods, *perceived* their dietary needs as incompatible with the preferences of other family members [and] found it unacceptably time-consuming to prepare two different meals, one for themselves and another for the rest of the family” (Zierner et al. 1996: 490-91, emphasis added). The authors concluded that “The issue of cost may be more perceived than real,” especially since all family members would benefit from a “prudent diet [and so] having separate meal plans for the patient and family should be unnecessary” (Zierner et al. 1996: 490-91).

These authors, it seems, dismissed patients’ reported concerns about lack of money to purchase better foods, lack of time to prepare different meals for different family members, and the desire to satisfy multiple family members (concerns that have been well documented and substantiated elsewhere (Devine et al. 2006)). Instead, they seemed to regard the problem as merely the lack of knowledge and willingness to make tough choices for other family members. Although these four articles comprise only a small percentage of the articles based on respondent self-reports, their dismissal of self-reports identifying poverty as an underlying cause of diabetes and its complications is too striking to ignore.

The de-linking of poverty from diabetes and its complications was further accomplished, in some cases, in the statistical analyses. Nine of the 30 articles in our sample statistically tested for the impact of income on rates of diabetes. Of these nine, four (44%) explicitly concluded that poverty had no impact. (One of these had also dismissed poverty by dismissing respondents’ descriptions of how poverty hindered self-care).

These four articles reached this conclusion by using a very narrow definition of poverty: under 100% of the federal poverty level (FPL). Researchers from the Urban Institute report that at incomes up to 200% FPL most individuals routinely cannot meet basic needs, and risk serious debt or even destitution if they face any unexpected expenses or income losses (Acs and Nichols 2005). Similarly, families with incomes up to 200% FPL are as likely as those below 100% FPL to report difficulty paying for food, housing, and health care (mostly because the latter receive considerably more government aid). For these reasons, most social scientists (including those at the Urban Institute) typically use under 200% FPL as their definition of poverty in statistical analyses.

In contrast, the authors of these four articles divided their samples into those living under the poverty line and those living between 100% and 200% FPL, identifying the former as “poor” and the latter as “non-poor.” Not surprisingly, these researchers found little difference in rates of diabetes or its complications between these two groups, since both groups were actually quite poor. Yet these articles made no mention of the limitations of their samples or data analysis, and

explicitly stated that poverty was not correlated with diabetes or its complications. In contrast, the remaining five articles that statistically explored the relationship between diabetes and poverty either used more realistic definitions of poverty or acknowledged that their sample was too homogeneous to test the impact of income on diabetes.

Just as emphasizing individual choices shifted attention away from poverty in the bulk of these articles, so, too, did an emphasis on ethnicity. This emphasis took various forms. First, six articles identified minority culture as the *primary* cause of diabetes. For example, articles argued that minority culture (rather than poverty, previous experiences, or other factors) leads individuals to value sweet, fatty foods (Eldeirawi and Lipton 2003) or to consider overweight children “a sign of the family’s prosperity and well being” (Bradshaw 2002: 548). Another four articles (one-third of the sample) identified ethnicity (via minority culture, genes, or low education levels) as a contributor to diabetes or its complications (see Tables 1 and 2).

Second, the articles reinforced the link between ethnicity and diabetes through their choice of sample. Nineteen of the 30 articles (63%) were based solely on ethnic minorities. Of these, one each described Russian Jewish immigrants, Filipino-Americans, and Appalachian whites, while the rest described Hispanic or African American populations. These nineteen articles either excluded non-minorities from their samples or omitted the small number of sampled non-minorities from their analyses.

Given the disproportionate impact of diabetes on minority populations, that so many of the articles in our sample focus on minorities is understandable. The *lack* of attention to diabetes in poor *non-minority* communities, however, is less understandable and more problematic, especially since studies conducted in countries that, unlike the United States, collect data on disease prevalence by socioeconomic status (e.g. the Netherlands, the United Kingdom, Spain and Canada), indicate that rates are increasing disproportionately among all poor, irrespective of ethnicity (Mackenbach 1994; Schrijvers et al. 1998; Larranaga et al. 2005; All Parliamentary Group for Diabetes and Diabetes UK 2006; Rabi et al. 2006). Similarly, the incidence of diabetes is known to be increasing among poor Appalachian whites (Wright 2003). We are unaware of equivalent data for other groups of poor white Americans.

The paucity of articles on diabetes among poor whites could easily leave readers with the impression that poverty and diabetes are only an issue in minority communities. Indeed, 15 of the 19 articles based solely on poor minorities (79%) made no mention of how their findings might apply to poor whites or to non-poor, or better still, middle, upper middle, or upper class minorities. Such comparisons would have clarified the relative importance of poverty and ethnicity, as well as their combined effects. In addition, the paucity

of research on poor whites may reinforce the idea that something about whiteness protects poor whites and that something about minorities themselves, independent of poverty, is a primary cause of diabetes and its complications. This idea is illustrated by Hosler Melnik, and Spence (2004) who wrote: “Despite their White racial background, the prevalence of diabetes in Russian-speaking immigrants was high, and comparable to the rates of other non-White groups known to be at risk for diabetes,” before going on to discuss how cultural factors place non-White groups at high risk (p. 375). The phrase *despite their White racial background* suggests that finding high rates of diabetes among these whites surprised the authors by violating their expectation that whiteness itself is somehow protective.

Surprisingly, given the current emphasis in medicine on genetics, the topic received little discussion in the articles we surveyed. Six articles mentioned that genetics contributed to diabetes and its complications, but none identified it as the primary cause (Tables 1 and 2).

PROPOSING SOLUTIONS

To understand how these articles conceptualized potential solutions to the diabetes epidemic, we first identified each locus of change that was recommended in one or more articles, and collapsed this list into five categories: individual patients, providers, health care access, economic inequality, and genetics. We then coded each article, identifying *all* loci of change that the authors suggested were important and, where possible, identified the locus of change that they suggested was *most important* (Table 3).

Table 3: Loci For Change Identified as Important or Most Important in 30 Articles on Diabetes among the Poor.

Locus for Change	Locus considered important*		Locus considered most important**	
	N	%	N	%
Patient attitudes or behaviors	25	83%	21	70%
Health care provider attitudes or behaviors	10	33	3	10
Health care access	9	30	3	10
Economic inequality	2	7	1	3
Genetics	0	0	0	0
Multiple or unclear answers	NA		2	7
Total	NA		30	100%

* Answers are *not* mutually exclusive

** Answers *are* mutually exclusive

Given that the U.S.-based articles most commonly identified individual choices as the primary cause of diabetes and its complications, it is not surprising that discussions of solutions also focused primarily on changing patients' beliefs, attitudes or behaviors. The overwhelming majority of the articles (83%, $n=25$) implicitly or explicitly recommended research or education to change patients. Articles recommended such strategies as teaching patients to more fully understand the dangers of diabetes, helping them develop healthier versions of traditional recipes, or "empowering" them to take charge of their diabetes. Even articles that showed great sensitivity to the plight of poor patients typically ended with calls to work harder to change patients' beliefs, attitudes, or behaviors, rather than to change their economic circumstances that may well have led to disease-promoting beliefs, attitudes or behaviors in the first place. For example, after describing in detail how "resource limitations of time [and] money ... introduce important barriers to self-care," Hunt et al. concluded not by calling for redistributing monetary resources in any way, but by suggesting that "providers and patients together...generate creative solutions to resource problems, [such as] developing strategies for controlling food costs within a healthy diet" (Hunt, Pugh, and Valenzuela 1998: 209).

In contrast, only one-third of the articles ($n=10$) recommended educating providers (half promoting greater "cultural competence" and half promoting further education on diabetes or interpersonal skills). Similarly, just under one-third ($n=9$) recommended improving health care access (typically limited to improving access to a specific educational program or diagnostic procedure such as annual eye exams). Only two of the articles (7%)—one published in a social science journal and one in a public health journal—suggested reducing diabetes and its complications by reducing poverty.

Similarly, 21 of the 30 articles (70%) concluded that the *most* important locus for change was the individual patient, three articles (10%) focused on changing practitioners, and three (10%) on changing the health care system. Only one article (3%) recommended reducing poverty as the *most* important means of reducing diabetes and its complications. The remaining two suggested more than one primary locus of change. As might be expected, given the limited discussion of genetics as a cause of diabetes in these articles, no article identified genetics as a primary or recommended locus for intervention.

DISCUSSION

Substantial data collected over numerous studies in multiple settings indicate an association between poverty and diabetes, and there is compelling evidence to believe that the association is causal, i.e., that poverty status, through a range of mechanisms, leads to biological processes that may result in diabetes or its

complications. Yet only a minuscule fraction of U.S.-based articles in the health care literature identify poverty as the proper target of interventions, and even when they do, they treat it, conceptually and analytically, as merely one individual risk factor that can be parceled out to allow for the study of more tangible, or targetable, risk factors—psychological, behavioral, cultural. The invisibility of poverty or its treatment as an individual failing leads even so-called public health strategies to rely on approaches that in the last instance “fix” disease states one individual at a time—albeit on a grand-scale.

Turning poverty into an “elephant in the room”—or, in this case, in the scientific literature—was accomplished in these articles through three main rhetorical mechanisms. First, the articles as a group emphasized the culture, ignorance, or psychology of the poor, while downplaying how poverty leads to those circumstances. Seven of the 30 articles further sidelined the impact of poverty by dismissing their subjects’ reports of the barriers it posed, or by using an overly-narrow definition of poverty to “demonstrate” that it was not causally related to diabetes or diabetic complications.

Second, a significant minority of the articles sidestepped the impact of poverty on diabetes by instead emphasizing ethnicity. Articles disproportionately investigated poor *minority* populations, rarely asked whether their findings applied to either poor whites or non-poor minorities, and typically suggested that ethnicity (via lower educational levels, genetics, or cultural attitudes and behaviors) directly led to diabetes or its complications. Only a fraction of these articles considered whether the more important factor might be the racism, political disenfranchisement, and material deprivation that frequently accompanies minority ethnicity and that can cause fatalistic attitudes, low education levels, and, in the end, ill health (Williams, Neighbors and Jackson 2003).

The final rhetorical move that downplayed the causal role of poverty was to virtually ignore it when proposing solutions to the growing diabetes epidemic — even when the authors recognized poverty as a major cause of diabetes. Instead of calls for reducing poverty itself, the articles assumed an unchangeable socioeconomic hierarchy and overwhelmingly called for interventions to help patients “adapt” to it by increasing their knowledge of diabetes, empowering them to adopt diabetes-preventing behaviors, or by “tailoring” or “targeting” educational material to their allegedly special belief systems or limited cognition.

These calls were well meaning, and often recognized the need for interventions to be “culturally sensitive.” But by identifying individuals or social groups as “problematic” and focusing on individual-level solutions, these articles implicitly suggested that these individuals or communities can be fully empowered to choose healthy lifestyles, regardless of social, institutional or political arrangements that make such lifestyles virtually unattainable. By

extension, the articles implied that broader societal changes, such as providing equitable access to medical care, protecting U.S. jobs, or mandating living wages, are unnecessary to improve public health.

Of course, few of these articles were *designed* to directly address the link between poverty and diabetes, and we do not intend to fault individual authors for centering their research and writing on the questions they find most pressing. It is not surprising that, for example, an article on nutrition education for poor persons with diabetes would focus on describing educational programs and their effects. On the other hand, given the great impact that poverty has on nutrition, it is reasonable to expect authors, in describing these programs, to discuss how poverty affects individuals' nutritional options, ability to attend educational programs, and ability to use the materials presented therein. Authors *choose* whether or not to do so. We argue that the *absence* of such discussion is as important as its presence. At the very least, it is likely to influence what the larger medical community concludes is the "problem," what that community communicates to the public, and what the public accepts and ultimately demands from health care practitioners, policy makers and politicians.

What do these findings tell us about the current state of diabetes prevention, research and care in the United States? And what are their implications for reducing health inequalities more broadly? Individual researchers, of course, have every right to focus their research on the topics they find most compelling. Our research suggests, however, that these individual choices are resulting in a serious gap in the literature. More importantly, given the social prestige of scientists, these choices may contribute, however inadvertently, to public complacency *vis-à-vis* socioeconomic inequities, by making the health inequalities that ensue appear legitimate or inevitable.

The role of poverty and social inequality in disease causation has been suspected, if not known, for at least two hundred years. In 1790, the "father" of public health, Johann Frank, in his address to young physicians, asserted that the "Mother of Diseases" was the "people's misery" (in other words, their poverty) (Frank 2003/1790). Since then, the top causes of disease and death have shifted dramatically, from acute infectious diseases to chronic diseases. However, the link between health status and social status is confirmed in study after study (Chaturvedi et al. 1998; Evans et al. 2000; Bachman al. 2003; Forssas et al. 2003; Green et al. 2003). And while it has become fashionable to explain mortality and mortality differentials as a "complex interplay of genetic, biological, social, environmental, cultural and behavioral factors, the "people's misery" remains the major cause of ill-health, and social class the strongest predictor of health differentials (Davey Smith and Egger 1993: 1086).

One compelling explanation for this unchanging relationship is Link and Phelan's theory of "fundamental social causes" of health and disease (Phelan

2004). This theory proposes that, across time and across diseases, money, knowledge, prestige, and beneficial social connections allow wealthier individuals to protect or restore their health by utilizing whatever resources are available in that place and time. As a result, social disparities increase when conditions are highly preventable or curable (such as ischemic heart disease and pneumonia) and diminish when conditions are neither preventable nor curable (such as pancreatic and prostate cancer), leaving even the wealthy without effective options (Phelan and Link 2005). This may explain why, as knowledge about how to prevent and treat disease developed during the twentieth century, diabetes and heart disease came to disproportionately affect ethno-racial minorities and the poor.

Why, then, would health researchers downplay or ignore how poverty leads to diabetes and, even more obviously, to its complications? One likely explanation is that in the United States as elsewhere, the culture and structure of medical training and, to a lesser extent, of other health-related fields, typically stresses individual-level factors in the etiology, prevention, and treatment of disease (Mishler 1981; Martin 1987; Waitzkin 1989). This would explain why individual-level explanations were more common in clinical medical journals than in those that took a more public health or social science approach.

A second possible explanation is that, as Leonard Syme argues, addressing social inequalities seems “too daunting” (Syme 1998:493). As a result, Syme suggests, “typical epidemiological research...holds social class constant...so that the importance of *other* factors can be assessed” (Syme 1998:493). Similarly, other health researchers may conclude that it is most practical to focus on achieving individual incremental changes rather than on broader social changes. Although this tendency is understandable, shifting the focus of inquiry and intervention from poverty to other factors—or alternatively, treating poverty as merely an individual trait rather than as the product of social organization—obscures our understanding of disease etiology and processes and undermines our ability to improve the health of the population.

Peculiarly American cultural tendencies may also partly explain the disconnect between poverty and illness in health literature on diabetes. American culture in general stresses individuals’ ability to shape their own destinies and holds them responsible for their successes and failures (financial and otherwise). This tendency leads to “blaming the victim” rather than to searching for social causes and social solutions to social problems (Zola 1972; Crawford 1979; Tesh 1988; Weitz 1991), and may partially explain why even articles in public health and social science journals rarely called for “targeting” social and economic policies leading to poverty directly.

At the same time, partly because American society is so obviously divided by race, American popular culture typically downplays class divisions and paints an

image of the United States as a “middle-class society.” One consequence of this is that in American political culture and activism, concern over socioeconomic inequality is often subsumed into concern over racial inequality as, for example, in affirmative action programs that aid individuals based on ethnicity with no attention to social class (Zweig 2001: 21). It is not surprising that a similar pattern occurs in American health care culture, research, and policy.

Whatever the reasons for the systematic downplaying of poverty in expert literature on diabetes, and whether intentional or unintentional, it serves a social function. As Zerubavel (2006) has suggested, public silences and denials always draw on culture-wide strategies of “mutual avoidance” which enable individuals and social groups to proceed with “business as usual.” In this case, medical silence over the impact of poverty on human health allows clinicians, researchers, policy makers, and the public in general to focus on individual-level causes of and responses to illness, rather than on sociopolitical ones. By so doing, this silence allows those within the health care world to stay in their comfort zone, using their accustomed tools, theories, and worldviews, and allows all of us who benefit from social inequality to avoid asking whether and how our privileges are linked to others’ suffering.

Our study has limitations: for one, we did not conduct in-depth interviews of the authors of the articles examined. Doing so might have allowed us to explore further these authors’ thoughts concerning the power inequalities underlying the poor health of their research subjects, and to better understand the rationale for the topics they chose or how they framed their results. Evidence suggests, however, that this lack of concern for how social power shapes health inequalities is part of the professional ethos of medical practitioners, and that public health practitioners have adopted this ethos in hopes that a more “scientific” and “objective” stance will improve their profession’s social status (Brandt and Gardner 2000).

Another limitation is that our study only included US medical settings, and therefore is not generalizable to other countries with different medical and political cultures. However, there is evidence that the individualization of social problems as they affect human health pervades non-American settings as well, even in disciplines with a greater self-awareness of the structural determinants of health and disease than clinical medicine, such as social medicine (Porter 2006).

Yet another limitation is that we only looked at thirty articles among the thousands published on type 2 diabetes, and only those that were accessible to us through the database of the National Library of Medicine. Yet the paucity of articles identified through main subject headings as concerned with poverty is itself revealing, especially considering the importance of the National Library of Medicine as a leading and authoritative institution in health and health care matters, not only in the United States but worldwide. Notably, this paucity

mirrors the results of a search conducted by one of the authors in the research database of the American Diabetes Association, the largest non-profit in America involved in diabetes prevention and treatment. This search retrieved not a single project concerned with diabetes and poverty (Chaufan 2006). We find these trends and silences worth highlighting and investigating further.

In any event, the point of our work was not to quantify unquantifiables, i.e. silence and invisibility. Rather, our goal was to show how the community that arguably has the greatest jurisdiction upon matters of health, namely, academic health researchers, treated a well-established cause of diabetes discursively. We intended to identify trends in what is being said and (perhaps most importantly) what is *not* being said, and to theorize about the implications of these discursive practices both for individual health and for the health of the population. Health care workers who are not trained to recognize the impact of poverty will be less able to assess whether their prescriptions—for drugs, diet, or behavior—are realistic for poor patients. When their patients subsequently fail to comply, or fail to improve despite complying as best they can, these health care workers will likely assume that patients have *refused* to comply or, more sympathetically, *cannot* comply for reasons pertaining to idiosyncratic cultures or belief systems. Meanwhile, when health care providers don't understand how poverty narrows patients' options, their patients will either be less likely to trust the advice and prescriptions they receive or more likely to blame themselves unnecessarily for their failure to improve or to comply. Either way, relationships between patients and providers are likely to deteriorate, along with patients' sense of self-efficacy. This sequence leads care providers to observe, correctly, that low self-efficacy correlates with poor diabetes control, but to conclude, *incorrectly*, that patients' psyches, rather than their poverty, is the most fundamental problem.

At a broader level, the silence surrounding the causal role of poverty in diabetes encourages American policy makers, politicians, and citizens to assign "blame" for the diabetes epidemic to the "peculiar" biology, behaviors, psyches, or cultural practices of persons with diabetes. This in turn shifts discussion and policy towards individualistic patches (such as "empowering" the poor to "take charge" of their health destinies) that can offer at best incremental improvement, while ignoring potential social changes (such as mandating living wages, guaranteeing affordable housing, or granting a social right to health care) that could dramatically improve the public's health.

To truly improve population health in America, health researchers who examine diabetes and other illnesses that disproportionately affect the poor should test theories as well as programs that acknowledge poverty's role in disease causation and should use this knowledge to advocate for transforming structural conditions at their root. Importantly, they should acknowledge the power inequalities that produce and reproduce poverty status in specific

segments of the population, i.e., the politics of poverty, and make explicit whether they understand poverty to be caused by “individual or community failings” or by “public policies that create unjust and inequitable distribution of economic and social resources” (Raphael 2007:13), our preferred explanation.

Clearly, interventions likely to spring from either causal analysis will frame the problem of, and the solutions to, ill health and health inequalities in radically different ways. Indeed, the politics of poverty is an important area of inquiry, albeit under-explored. There is reason to believe that politics, policies and population health are causally and hierarchically related; that is, it is not population health status that leads to specific policies and political traditions but the other way round. One study, for instance, makes a persuasive case that social democratic countries with a stronger tradition of labor unions have substantially better health indices, presumably via redistributive social and economic policies (Navarro and Shi 2001). If this relationship is further substantiated, a logical step for researchers advocating for decreasing health disparities and improving health overall would be to support social movements and political parties that are fighting for political forces such as the labor movement and social democratic parties, which have traditionally backed larger, more successful redistributive policies (Navarro and Shi 2001: 490). Meanwhile, social science researchers should focus on how downplaying the link between poverty and illness influences the nature, development, and consequences of diabetes and other poverty-linked illnesses at individual and societal levels. They should also explore how the current social constructions of the diabetes epidemic and of other poverty-linked epidemics developed. In particular, they should investigate who benefits and who loses from the way these problems are currently framed and how we can reframe them to reveal their socio-political roots and support socio-political solutions.

As George Lakoff writes (2002:xv): “Frames are mental structures that shape the way we see the world...the goals we seek [and] the plans we make,” so changing frames is a crucial step in creating meaningful social change. Only with a better understanding of the processes that produce and maintain the invisibility of poverty in research on type 2 diabetes and other illnesses will we be able to truly stem “modern” epidemics, including those of chronic diseases, and, more generally, reduce health inequalities.

ENDNOTES

¹PubMed is the public access version of Medline, and indexes the same journals with the same search terms and strategies; both databases were searched to confirm this.

²In developing nations, poverty is more obviously life threatening, and in European countries, all citizens have at least basic health care access

³List of articles available upon request.

⁴Code-sheet available upon request.

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