

Figuring Out Type 2 Diabetes through Genetic Research: Reckoning Kinship and the Origins of Sickness

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Genetic research on type 2 diabetes serves as a point of departure in this paper. Drawing together classic work in the anthropology of medical epistemologies and the recent revitalization of kinship studies, the paper has two main objectives: (1) further unsettling a portrait of biomedicine as having a single overarching epistemological orientation that locates the origins of disease squarely within individual human bodies; and (2) inviting further reflection and discussion about history, social structures and cultural norms as bona fide causes of disease. The paper shows that causal roles ascribed to history, social structures and cultural norms through genetic research on diabetes hinge on underscoring evolutionary 'blood relations' between people, as well as between human and 'lower' nonhuman beings. It is argued that type 2 diabetes has not thoroughly undergone geneticization, but, partly through genetic research, it has undergone greater medicalization. Despite broad consensus that 'the environment' is the root cause of increased type 2 diabetes incidence, proposed remedies still tend to privilege clinical management.

Introduction

What relationships are emphasized and brought into being through contemporary genetic research on diabetes? Some have argued that research has geneticized diabetes (McDermott 1998; Hedgecoe 2002). The term 'geneticization' was coined by Lippman (1991) to extend the concept of 'medicalization', the framing of social problems as individual pathologies. Hedgecoe (2002, p. 8) argues that social scientists can and should assess whether geneticization has actually taken place, and

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offers a definition that does not pivot on a moral stance: ‘in medicine, geneticization takes place when a condition is linked to a specific stretch of DNA’. The present paper applies this definition, and finds that while type 2 diabetes has not thoroughly undergone geneticization, it has undergone increased medicalization.

Two objectives thread through this paper. First, this paper seeks to unsettle further a portrait of biomedicine as having a single overarching epistemological orientation that locates the origins of disease squarely within individual bodies. Second, this paper is intended to invite further discussion about the implications of recognizing history, social structures and cultural norms as *bona fide* causes of disease. In the end, sociocultural research on biomedical knowledge cannot elide questions about the *objects* of biomedical knowledge, particularly when social organization and cultural norms are understood to play a role in shaping these objects. Type 2 diabetes is a strategic example for making such arguments, because anthropological accounts of type 2 diabetes causation privilege history, social structures, and cultural norms (Szathmary 1994; Young 1994; Gittelsohn *et al.* 1996a, 1996b, 1998; Young *et al.* 2000; Benyshek *et al.* 2001; Rock 2003).

Analytically, the paper builds on the recent ‘revitalization’ of kinship (Strathern 1992 in particular; Haraway 1997; Franklin 2001), by way of an examination of the fit between genetic research on type 2 diabetes with two classic papers in the anthropology of medical epistemology (see Young 1976; Foster 1998 [1976]). Empirically, the focus is a public presentation¹ given on 13 October 2000 as part of the Canadian Diabetes Association Professional Conference held that year in Halifax, Nova Scotia, Canada. ‘The Search for Diabetes Genes: Where Have We Been and Where Are We Going?’ was the title of this plenary session. Some 1,700 professionals (physicians, nurses, dieticians, social workers, and pharmacists) hailing from across Canada were on hand to hear this presentation. The keynote speaker, Dr Morris Birnbaum, touched on type 1 diabetes (formerly known as juvenile diabetes, or as insulin-dependent diabetes), but he mainly dealt with type 2 diabetes. His overview of genetic knowledge about diabetes, intended for an audience of health professionals, is germane for illustrating and reflecting on contemporary biomedical knowledge.

Types of Type 2 Diabetes

After briefly invoking type 1 diabetes to illustrate the concept of complex inheritance, which applies to conditions in which more than one gene is involved, the speaker outlined some methodological challenges in genetic research on type 2 diabetes. Unlike with type 1 diabetes, it was noted that no single region of the genome has been tied to type 2 diabetes. Instead, any number of gene combinations may be involved, and challenges arise because type 2 diabetes usually develops much later in the life than type 1 diabetes. Dr Birnbaum explained:

When you’re dealing with a disease which people [tend to] get late in life, doing these genetic studies can be very, very difficult, because how many generations could you really get back? Think to yourselves, how many of your patients can you ask,

'Does your grandfather have diabetes and will he come in to donate blood?' [laughter from audience]

In addition, it was noted that genetic researchers cannot be sure that siblings or even parents of people with type 2 diabetes will not ultimately develop the condition themselves. The bulk of the presentation reviewed examples of the methodological strategies employed by researchers to overcome these challenges, and some results that they have yielded.

From Mexican-American Families to Calpain-10 . . . to Diabetes

To circumvent the challenges inherent to researching adult-onset conditions, geneticists often employ the 'affected sib-pair analysis' approach:

Affected sib pairs only looks at siblings who have the disease. And it's based on the following principle: that if two siblings have the disease, they're more likely to have the same gene at the region that is causing the disease.

To illustrate the potential utility of sib-pair analysis for investigating type 2 diabetes, the speaker described results from research in a Mexican-American population:

Anyway, Graeme Bell and his colleagues at the University of Chicago over the last 10 or 15 years have been doing a study just like this . . . And through a very convoluted genetic argument that I couldn't, I couldn't explain now if I wanted to, they've really come up with the idea that a specific gene called calpain-10 is associated with diabetes in this population². . . But most importantly, before Graeme Bell identified this gene, there was *no reason* in the world to suspect that this protein had anything to do with diabetes. And this is the strength of this approach.

Dr Birnbaum thus underscored the laborious nature of this kind of research, but also its potential for generating new hypotheses about type 2 diabetes.

MODY: When Diabetes is an Unequivocal Inheritance

MODY (for 'maturity onset diabetes of the young') forms, while rare, could together account for many cases. 'Conservatively, it's at least 5% and in some populations, this figure may reach 20%', the speaker said. Current treatment recommendations do not differentiate MODY cases from what Dr Birnbaum called 'garden variety' type 2 diabetes. 'So right now it's not a major problem that you might not be able to distinguish these patients', he noted.

Unlike type 1 diabetes or 'garden variety' type 2 diabetes, MODY forms of diabetes are *not* genetically complex. The audience was told that with MODY forms, 'You have a 50% chance of getting it [diabetes] from an affected mother or father. . . If you have the gene, you almost invariably get the disease'. He illustrated heterogeneity in terms of severity and prognosis among MODY forms by profiling research carried out by two of his colleagues.

Geneticists found that a region of chromosome 7 contained what would become known as MODY-2 gene, but they could narrow it down no further until they drew upon physiological expertise: 'Franz Matschinsky at my institution, the University of

Pennsylvania, has argued for 25 years that glucokinase is the protein that is the key to insulin secretion'. Further research linked a particular mutation in the glucokinase gene to MODY-2, which has distinct clinical features:

You get hyperglycemia early in childhood . . . even before [age] 10. The hyperglycemia is mild, and it doesn't really change with age. Where you are early in life tends to be where you're going to be when you're grown up. There's very little in the way of complications. It's unclear right now whether there's any reason to treat this disease. The glucose is up, but it's a mild hyperglycemia.

Dr Birnbaum then contrasted this genotype with MODY-4.

When another colleague at the University of Pennsylvania encountered a baby born without a pancreas, it was already known from experiments on mice that two copies of the gene PDX results in the congenital absence of a pancreas.

Well, Doris Stoffers, in looking very carefully at the relatives [of this baby], noticed something very interesting. She noticed that the father had diabetes. No big deal, it's a common disease. But even though he had what looked like type 2 diabetes, he developed it when he was 17 years old. OK. And she couldn't find any evidence of an immune response, indicating this wasn't type 1 diabetes. 17 years old with type 2 diabetes, OK. So she looked at the larger family and developed a pedigree, and looked at all of the individuals in the family who developed diabetes at an early age.

The pedigree showed that about half of the baby's paternal *and* maternal relatives had a copy of the PDX gene and developed type 2 diabetes in early adulthood. 'But it's clearly very different from garden variety type 2 diabetes', Dr Birnbaum emphasized, because it is associated with one specific gene and those who inherit two copies of the gene are born without a pancreas.

People with the same MODY forms are felt to have similar 'life chances' because they have a powerful genetic substance in common, one that necessarily causes diabetes. By contrast, in 'garden variety' type 2 diabetes, contemporary genetic research has attributed a pivotal role to the surrounding 'environment', as sufficient to cause many cases. The sharing of substance, of elevated blood glucose levels but not necessarily of genes, is partially attributed to shared or comparable lived experience in cases of 'garden variety' type 2 diabetes.

Extrapolating from the 'Current Dogma'

Contemporary genetic research may begin with affected people, but it often 'start[s] from the biology', explained the speaker. He reminded the crowd that expertise about normal physiology was instrumental in characterizing MODY-2. 'The current dogma' about the physiology of type 2 diabetes was summarized as follows:

So in type 2 diabetes, we have three defects: an inability of the pancreas to respond appropriately to the glucose; an inability of the liver to stop making it; and an inability of the muscle to take it up appropriately.

He then presented on research from his laboratory, on whether a 'normal protein' known as AKT may play a role in diabetes onset. Once genetically programmed to

over-express AKT, mice were found to end up with eight to ten times more insulin-producing cells. The mice did not develop diabetes, even after being injected with a toxin that usually kills the insulin-producing cells. ‘We’ve completely prevented diabetes in these mice’, Dr Birnbaum underlined. But AKT over-expression would not be a practical therapy for people, he added, because the mice go on to develop other health problems.

Of Genes and Volition

The speaker repeatedly emphasized that genetic research may help researchers identify pathways never before imagined as linked to type 2 diabetes. This theme animated his presentation of research linking calpain-10 to diabetes in a Mexican-American population. In another example drawn from his own laboratory, Dr Birnbaum explained that depriving mice of a certain enzyme by ‘taking a very specific gene, and [introducing] a very modest modification’ reduced the desire to exercise:

What we’ve done here is we’ve taken mice, normal mice and mice which are deficient in this critical enzyme AMP-K in the muscle and the heart. And we’ve put them in a cage, put them in their cages, and we’ve put exercise wheels in the cages. Now we did not force them exercise. We’ve simply given them the opportunity to exercise.

Audience members laughed uproariously, prompting the speaker to say, ‘You’re way ahead of me here. I was going to draw the obvious analogies; I’m delighted you’re getting them long before I have to tell them to you’. Note that in this case, the modelling of type 2 diabetes in people encompasses a surrounding environment that allows for physical activity. The connection drawn by many audience members, which made them laugh out loud, is with the social and physical environment that surrounds *them*.

Modelling Origins, Invoking Histories: Drosophila to the Rescue?

The speaker concluded his lecture by suggesting that the study of organisms ‘lower’ still than mice may yield genes linked to the onset of type 2 diabetes in humans. To introduce this approach, he suggested what an ideal diabetes gene-hunting expedition might look like:

We do a fasting glucose [test] on 100,000 mice, repeat it three times because it’s pretty variable, find the mice that have diabetes, and clone out the genes. Identify the region of the gene that causes the diabetes, and figure out what it is. I mean, that’d be a great experiment. We’d probably find a lot of diabetes genes that way.

He paused for dramatic effect, and then he broke some bad news: ‘That experiment is technologically, financially and emotionally impossible’. The audience roared with laughter. The keynote speaker continued, ‘I mean, none of us can *dream* of managing 100,000 mice’. The laughter, which had faded, resumed. ‘Now we’ve got to do 100,000, because it’s pointless to do this experiment unless we mutate

most of their genes', Dr Birnbaum said, or the likelihood of inducing a genetic mutation that is pertinent to the development of diabetes in humans would remain too low.

Next he introduced that most convenient of laboratory creatures, venerable *Drosophila*, as a possible key to understanding genetic contributions to diabetes in human beings:

On the other hand, there are organisms you can do that with. I'm just going to repeat: there are organisms with which scientists do that all the time. Yeast. Fruit flies are the classic organism, where you can mutate a fruit fly and screen 100,000 for ones that are missing wings, or have an extra eye, or, you know, do stuff that you look at under a microscope in 10 seconds.

The only problem is being sure that the findings have relevance for human beings. Yet fruit flies do produce insulin, and this commonality may permit fruit flies to stand in for human beings in genetic research on diabetes.

To demonstrate the potential viability of using fruit flies as model organisms for type 2 diabetes in people, Dr Birnbaum described research in his own laboratory and in a Swiss laboratory. These experiments show that in *Drosophila*, the insulin-signalling pathway regulates the size of the cell, the size of the wing and the size of the fly itself. Many members of the audience laughed out loud when Dr Birnbaum said, 'Now that's parallel in some ways with human beings'.

He continued:

I mean, if you just stop and pause for a second, and think about, you know, the fruit fly on your banana. And how unrelated he is to you. But to think that that fly uses insulin-signalling in the same way you do. It uses that insulin pathway to tell it that there's plenty of food around. . . Insulin-signalling *evolved* as a way of telling the organism that there's food around. What's changed a little bit in evolution is how the organism responds to the knowledge that there's a lot of food around. In a fly, it makes a bigger fly. In us, it takes the nutrients and converts it into . . . glycogen or triglyceride, or protein. But fundamentally, the response is the same.

Does the response, the larger response still exist in human beings? Well I think all of you know the answer. Think about your patients. Think about your infants of diabetic mothers. What's the single most impressive characteristic of those babies? Macrosomia. These babies are enormous. And they're not enormous just because they're storing more nutrients, no. They're enormous because the cells are larger. There's probably more cells also.

To wrap up this portion of his presentation, he said:

So we're very, very excited about this. Because now, we can take a fly with a large eye, a large eye because it has had an increase in insulin-signalling. And induce a mutagenesis: make mutations in 100,000 of those flies, and simply by brushing them under a microscope in a course of a year—still a year's work, but that's not bad—in a year, we can find other mutations that make that eye bigger, make it smaller. And those will be mutants in other parts of the insulin-signalling pathway. Mutations in genes that we currently *don't* know about. And then once we know about those genes in the flies, we'll look at them in the human, and figure out whether they cause diabetes.

Sustained applause followed the conclusion Dr Birnbaum's lecture. After the applause died down, the chair of the session said, in awe:

Wow. That was an absolutely outstanding talk, and a brilliant example of deductive reasoning. And there are actually not many people around, let me tell you, there are not many people around who can take highly esoteric, very scientific work and make it understandable to all of us. In fact, there are probably only one or two people around who can do that, and we have one of them here. So thank you very, very much for that.

Yet more sustained applause followed the chair's remarks. What initially seemed preposterous to the members of the audience—that human beings sufficiently resemble fruit flies for *Drosophila* to generate new knowledge about the origins of type 2 diabetes—seemed sensible to them by the end of his presentation. What is more, this audience enjoyed and appreciated the opportunity to see themselves as akin to *Drosophila*, while manifestly so different.

Inside Out and Outside In

Indeterminacy about phenotype, or rather what phenotype will become, has channelled genetic research into type 2 diabetes away from the study of 'family trees' in the usual sense of a pedigree, and towards the definition and study of populations whose members are dispersed across space and over time. This state of affairs informs the use of coeval creatures, such as mice or even fruit flies, as models for diabetes in human populations (cf. Fabian 1983). And in anthropology and social science more generally, this state of affairs carries implications for theories of kinship, sickness and responses to sickness.

The notion that all people and indeed all organisms are related if the lines of descent are traced far enough back has a matter-of-fact quality in genetic research that is at odds with how people in Western countries often think about kinship (Strathern 1996, pp. 529–530; Carsten 2001). Indeed, the health professionals on hand to hear Dr Birnbaum lecture on genetic research into type 2 diabetes on 13 October 2000 were surprised when asked to think about fruit flies as model organism for human diabetes. Nevertheless, ambivalence is arguably inherent to kinship reckoning (Peletz 2001). When fruit flies or mice stand in for diabetics in genetic research, the 'evolutionary distance' between nonhuman and human beings is both collapsed and utilized (see also Haraway 1997 on oncomouseTM). Genetic research that deploys nonhuman beings as model organisms for human diabetes does not create forms of kinship 'beyond blood', however, for blood glucose remains the central organizing concern (cf. Haraway 1997, p. 265; Franklin 2001, pp. 315–317). If 'blood is thicker than water', genetic research leads to a kind of 'clotting', such that kith thicken to kin.

The concern manifest in modern-day genetic research on diabetes with the origins of disease, whether genetic or environmental or some quantifiable combination of the two, qualifies the supposition that biomedicine reduces the origins of disease to observable or measurable qualities of individual bodies. This supposition underlies the medicalization thesis (Zola 1975; Lock & Gordon 1988; Ilich 1990 [1974]), and also the geneticization thesis (Lippman 1991). Hedgecoe (2002) argues that diabetes

has undergone geneticization because the condition has been linked to a specific stretch of DNA. His investigation focuses on type 1 diabetes and how it became distinguished from type 2 diabetes mainly on genetic grounds. But the condition known as type 2 diabetes has never been linked to a specific stretch of DNA. Indeed, only a minority of all cases has been linked to specific stretches of DNA, the stretches of DNA vary across these cases, and genes are not portrayed as the ultimate cause. Thus, type 2 diabetes has not thoroughly undergone geneticization.

Hedgecoe (2002) and also Lippman (1991) contend that, for geneticization to occur, causality need not be reduced to genes alone, while Lippman (1991) in particular argues that reducing causation strictly to genes is tantamount to medicalization. Yet extensive geneticization is not necessarily synonymous with extensive medicalization, because some instances of geneticization may give rise to interventions targeting root causes of disease that operate outside the bodies of sick people, versus clinical management. Still, as in the case of type 2 diabetes, partial geneticization may intensify medicalization, through efforts to predict and control disease processes internal to people's bodies.

To ground these arguments, it may be helpful to refer to one of the first efforts to theorize a preoccupation in biomedicine with processes internal to individual bodies. In this classic article, Young (1976) observed that, historically and cross-culturally, medical systems have varied in the extent to which significant events are thought to take place inside, as opposed to outside, the sick person's body. Young observed that *internalizing* systems tend to rely on physiological explanations, in which images and analogies are used to model events within the sick person's body; by contrast, *externalizing* systems tend to rely on etiological explanations that identify a point in time before which it is unnecessary to search for causes. Internalizing and externalizing explanations may be proposed in turn or in parallel for the same case of sickness, which provides the basis for pluralistic medical systems featuring explanations and treatments deriving from more than one epistemological tradition. Both physiological and etiological explanations concern the origins of sickness, but Young (1976) notes that physiological explanations focus on mechanisms, while etiological explanations focus on root causes.

To appreciate its innovation and continued importance for theorizing how people construe the origins of sickness, Young's (1976) taxonomy can be fruitfully compared to another classic approach to distinguishing medical systems introduced that same year, 1976. According to Foster (1998 [1976]), non-Western medical systems vary in the extent to which they invoke personalistic versus naturalistic causes. Foster's (1998 [1976]) understanding of personalistic medical systems closely resembles externalizing systems described by Young (1976). Foster describes personalistic medical systems as follows:

A personalistic medical system is one in which disease is explained as due to the *active, purposeful intervention of an agent*, who may be human (a witch or sorcerer), nonhuman (a ghost, an ancestor, an evil spirit), or supernatural (a deity or other very powerful being) . . . Personalistic causality leaves little room for accident or chance . . . (Foster 1998 [1976])

Similarly, Young says:

Externalizing systems concentrate on making etiological explanations for serious illness. Here, pathogenic agencies are usually purposive and often human or anthromorphized... Often only gross symptomatic distinctions are made, since the intrasomatic link between etiological events and sequences of biophysical signs is either ignored or not elaborated. (Young 1976, p. 148)

Foster thus focuses on the highly restricted role allocated to chance in personalistic systems, while Young is at pains to situate the sick body in externalizing systems.

Foster's characterization of naturalistic medical systems diverges from Young's characterization of internalizing medical systems, although at first glance, they too appear similar. Foster writes:

In contrast to personalistic systems, naturalistic systems explain illness in impersonal, systematic terms. Disease is thought to stem, not from the machinations of an angry being, but rather from such *natural forces or conditions* as cold, heat, winds, dampness, and, above all, by an upset in the balance of the basic body elements. (Foster 1998 [1976])

Here is how Young characterizes internalizing systems:

In internalizing systems physiological explanations are indispensable for organizing medical strategies... Western medicine, which is one instance of this type... concentrates on micro-level processes organized according to highly elaborated machine models. (Young 1976, p. 148)

Young thus explicitly theorizes biomedicine, while Foster excludes biomedicine from scrutiny.

Current biomedical knowledge about diabetes confirms Young's chief observation about variation in explanations for human sickness, namely, that explanations differ in whether they attribute the origins of sickness to events *inside* or *outside* sick bodies. Nevertheless, the materials presented in this paper suggest that, ultimately, it is impossible to rank accounts of the origins of sickness by the extent to which they focus on external causes. *Internalizing* accounts may vary in the number of body parts implicated and the degree to which these different parts are thought to interconnect. Using the individual human body as a unit of analysis, analysts can rank the physiological complexity of different explanations. *Externalizing* explanations, however, can only be contrasted, not ranked (see Figure 1).

Biomedicine may generate the most internalizing accounts of the origins of disease ever known in human history (as noted by Young 1976, p. 158), yet biomedical knowledge does not always focus on the insides of individual sick human bodies. The 'clinical gaze' remains riveted the inner workings of individual bodies (Foucault 1973). Nevertheless, research often locates the origins of sickness *outside* sick bodies and well beyond the clinic (cf. Canguilhem 1966 [1943]). Genetic research resembles the attribution of 'supernatural' causes, in that attention is trained on the relationships in which a sick body is embedded, and which gave rise to that body.

Internal to the Sick BodyPhysiology (Mechanism)

Examples:

- Ayurvedic Medicine
- Biomedical Diagnosis, Treatment

External to Sick BodyEtiology (Cause)

Examples:

- Witchcraft, Magic, Spiritual Beings
- Genetic Research

Figure 1 Origins of Sickness Revisited.

Genetic models might *appear* to internalize to a greater degree than physiological models, because they burrow even deeper into the body, but appearances sometimes deceive. To explain the presence of certain genes and their effects in a given organism at a given time, genetic researchers inevitably invoke external phenomena. The external phenomena implicated by genetic researchers to account for the onset of diabetes have included genetic inheritance from one's parents at the moment of conception; evolutionary changes over time; and interactions, mediated by genotype, between the external environmental and the individual body. Just when the biomedical researcher's gaze seems directed to the very most inner reaches of the body—the preserve of genes—the focus of attention inverts: phenomena outside the sick human body become the focus of scrutiny. In other words, *this* biomedical gaze ultimately externalizes rather than internalizes.

Crucially, the recasting of kinship through the intermingling of internalizing *and* externalizing explanations for diabetes raises—without resolving—questions about responsibility. Since bodily action, biological endowment, evolutionary history and the organization of society all figure in ostensibly 'genetic' explanations, the onset of sickness appears *partially* subject to individual and collective will, including a will to overcome through further research. Specialists readily acknowledge that rising type 2 diabetes incidence mainly reflects 'environmental' conditions, yet the remedies proposed have tended to centre on clinical interventions (cf. Lloyd & Hawe 2003 on prenatal depression). Externalizing epistemologies generally tend to give rise to actions emphasizing change in the relationships embedding the sick person (Young 1976), but not so far in modern genetic research, given its strong links with clinical interventions that target individual bodies for change.

In recent years, type 2 diabetes has become subject to more intensive medicalization (Sinding 1999; Ferzacca 2000; Broom & Whittaker 2004; Rock 2004). Still, genes are not being fingered as the sole or main causes, and genetic technologies and services are not being construed as the only solution. Instead, it is envisioned that genetic knowledge could usefully inform clinical interventions. Indeed, this is already happening in some quarters, for example, in allocating kidney donations to recipients (Hogle 2000, pp. 102–103), many of whom are diabetic. In other cases, social interventions co-exist alongside clinical interventions, with both informed partly by genetic research, as in the Aboriginal community with the third-highest

type 2 diabetes prevalence ever reported (Hanley *et al.* 1995; Harris *et al.* 1997; Hegele *et al.* 2003).

Social researchers might want to argue in favour of more social interventions, given the acknowledged importance of 'the environment' in rising type 2 diabetes incidence (McKinlay & Marceau 2000). But should social structures and cultural orientations be deliberately manipulated to avert and control disease? And must medical anthropology serve medicine in the end? In thinking through these issues, it may be helpful to distinguish an anthropology for medicine from an anthropology for health and justice.

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Notes

- [1] Public presentations such as this at Canadian Diabetes Association Professional Conferences were among the key sources in my ethnographic investigation of how diabetes recently gained recognition as a public health and social problem. Cassette tapes of most sessions were available to the public because lecturers had signed a consent form permitting their sessions to be recorded. Not all lecturers, however, gave their consent and so not all sessions were recorded and sold. Upon request, the author will provide a copy of the standard consent form.
- [2] For discussion of the politics of this research, see Montoya (2001).

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