

Native Americans, Stress, and Type 2 Diabetes:
Exploring the Roots of the Epidemic

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Abstract: Type 2 diabetes mellitus is reaching epidemic proportions in many populations despite significant efforts and awareness in both the medical and lay communities. This article explores the proposition that the current diabetes etiological model of genetic predisposition and poor lifestyle choices is incomplete. It is suggested that the model be expanded to include both the underlying reasons for unhealthy behaviors as well as the direct physiologic effects of stress and trauma through the HPA axis and autonomic nervous system. This expanded model is looked at in the particular context of the population often most at risk for diabetes: Native Americans.

Native Americans (American Indians and Alaska Natives) have known many plagues since the Spanish first arrived in the mid-1500s. Smallpox, measles, typhus, typhoid, tuberculosis, cholera, scarlet fever and other diseases devastated Native populations. Today there is a new plague, type 2 diabetes, which affects all races but is disproportionately high in disadvantaged and minority populations. Native Americans as a whole have up to three times the rate of diabetes of non-Hispanic whites, but certain Indian groups have far higher rates still. The highest rates of type 2 diabetes in the world are in the Pima Indians of Arizona¹. However, diabetes is not the only plague in Indian Country. Dr. Michael H. Trujillo, Director of the Indian Health Service (IHS), states,

“The disparity of health status for American Indians and Alaska Natives has many underlying causes: Social and cultural disruption of traditional native societies, lack of education and economic opportunities and high levels of unemployment and poverty all put Indian people at higher risk. The consequence is disproportionately high levels of disease and health problems among Indian people.”^{2(p 2)}

Could the plagues of diabetes and poverty be related? Is it unreasonable to think that the collective and individual experiences of a people have a direct impact on their physical health?

The current model of diabetes holds that the disease is caused by a combination of genetic predisposition (“thrifty gene” in Native Americans) and unhealthy lifestyle choices (i.e. being sedentary and eating a high fat/calorie diet). Diabetes practice today reflects this model in its emphasis on educating patients on healthier lifestyles and using medications when these are not sufficient. It is evident this model is not complete,

however, as it has yielded few programs producing consistent risk factor reduction for significant numbers of people. Indeed, as Wing *et al* noted in one such study, “Although initially successful, the interventions studied (diet and exercise) here were not effective in producing long-term changes in behavior, weight, or physiological parameters. ...further research is needed to determine how best to increase the percentage of subjects achieving at least a modest weight loss.”^{3(p 350)} While obesity has become epidemic in the U.S. and across Indian Country, it is not because of a lack of awareness or effort: Serdula *et al* looked at Behavioral Risk Factor Surveillance System (BRFSS) data and found that 78% of women and 63.9% of men were actively attempting to either lose or maintain their weight .⁴

If people are generally aware of the major lifestyle changes needed to prevent or control diabetes, why has there been so little success in lifestyle modification? Or, in other words, why is education necessary, but definitely not sufficient, for true long-term health? As we try to find ways to translate the success of the Diabetes Prevention Program (NIDDK, Press Release August 2001) into programs for large numbers of people, answering these questions will be critical. Rozanski *et al* reviewed the literature on another facet of the Insulin Resistance Syndrome, cardiovascular disease, and concluded,

“Recent studies provide clear and convincing evidence that psychosocial factors contribute significantly to the pathogenesis and expression of coronary artery disease (CAD). This evidence is composed largely of data relating CAD risk to 5 specific domains: (1) depression, (2) anxiety, (3) personality factors and character traits, (4) social isolation, and (5) chronic life stress. Pathophysiological

mechanisms underlying the relationship between these entities and CAD can be divided into behavioral mechanisms, whereby psychosocial conditions contribute to a higher frequency of adverse behaviors, such as poor diet and smoking, and direct pathophysiological mechanisms, such as neuroendocrine and platelet activation.”^{5(p 2192)}

Perhaps we similarly need to expand the current diabetes model to include both, (1) the underlying reasons for “unhealthy” behavior choices as well as, (2) the direct effects of stress, poverty, low self-esteem, social isolation, and personal and community-wide trauma on diabetes risk—all of which cluster in lower socioeconomic status (SES) and minority groups, like Native Americans. The following sections will explore each of these two areas.

- (1) Health-related behaviors (e.g. overeating, smoking, sedentary lifestyle/excessive TV, alcohol abuse) are not primarily cognitively based; they are feeling based, stress-relieving responses.

A team from the CDC studied “Adverse Childhood Experiences” (defined as: emotional, physical, or sexual abuse; a battered mother; parental separation or divorce; growing up with a substance-abusing, mentally ill or incarcerated household member) and the risk for smoking (a known risk factor for diabetes) in over 9000 adults. Not only was the risk of smoking strongly associated with adverse childhood experiences, it was “dose dependent” with increasing likelihood of being a smoker with the more categories of abuse one experienced. The researchers went so far as to recommend that,

“Current smokers who consciously or unconsciously use nicotine as a pharmacological tool to alleviate the long-term emotional and psycho-biological

wounds of adverse childhood experiences may need special assistance to help them quit. Such assistance includes recognition of the use of nicotine to modulate problems with affect, treatment of the residua of these adverse childhood experiences, and the use of nicotine replacement therapy or antidepressant therapy.”^{6(pp1657-8)}

Similarly, data from the Kuopio Ischaemic Heart Disease Risk Factor Study were used to look at the association between unhealthy lifestyle choices and SES. Lynch et al comment that, “Results show that many adult behaviours and psychosocial dispositions detrimental to health are consistently related to poor childhood conditions, low levels of education, and blue-collar employment. ...Understanding that adult health behaviour and psychosocial orientations are associated with socioeconomic conditions throughout the lifecourse implies that efforts to reduce socioeconomic inequalities in health must recognize that economic policy is public health policy.”^{7(p 809)} Bessel van der Kolk addressed the mechanisms by which adverse childhood experiences affect behavior:

“Secure attachments with caregivers play a critical role in helping children develop a capacity to modulate physiological arousal. Loss of ability to regulate the intensity of feelings and impulses is possibly the most far-reaching effect of trauma and neglect. It has been shown that most abused and neglected children develop disorganized attachment patterns. The inability to modulate emotions gives rise to a range of behaviors that are best understood as attempts at self-regulation. These include aggression against others, self-destructive behavior, eating disorders, and substance abuse. The capacity to regulate internal states affects both self-definition and one’s attitude toward one’s surroundings.”^{8(p 145)}

Looking at eating behavior, Pine found,

“All obese and high-anxiety-condition subjects consumed more food than did nonobese and low-anxiety-condition obese subjects, respectively. The overall consumption of food was greater with American Indians than with White Americans...An alternative stress-reaction theory is proposed to more fully account for American Indian eating behavior. Obese and nonobese American Indians overeat in response to stress.”^{9(p 774)}

Or, as Redford Williams states, “Growing up in such conditions could teach the child of parents with lower SES that the world is a hostile, depressing, and alienating place, and the child could also learn that smoking and consumption of larger amounts of alcohol and food help reduce the resulting distress.”^{10(p 1746)}

Poverty plays another role in risk behaviors in that food insecure women are more likely to be obese than women who have a constant food supply.¹¹ Further, the types of foods which are both available and affordable in impoverished area are often the highly processed, high fat and simple carbohydrate foods most likely to contribute to obesity and diabetes risk.

In light of studies such as these, simply educating people on the adverse health consequences of their behaviors seems almost absurd when it is realized that these behaviors are actually strategies to help them cope with multiple life stressors. If diabetes risk behaviors like overeating and smoking are actually stress-relievers, then stopping them without healing the underlying stress issues only further increases the stress--which may help explain the high long-term failure rates of conventional behavior

change programs.

- (2) Stress and trauma have direct effects on diabetes risk through the Hypothalamic-Pituitary-Adrenal (HPA) axis and the autonomic nervous system.

The Healthy People 2000 initiative made the assumption that since both mortality and unhealthy behaviors are higher in lower SES people, it is the unhealthy behaviors which explains the higher mortality compared with high SES people. A group from the University of Michigan decided to test this assumption. They controlled for four behavioral risk factors (cigarette smoking, alcohol drinking, sedentary lifestyle, and relative body weight) in the 3617 adults participating in the Americans' Changing Lives survey. They found that these major behavioral risk factors only accounted for 12-13% of the difference in mortality. In considering what could be causing the "lion's share" of the mortality difference caused by SES, they state,

"Persons in lower socioeconomic strata have increased exposure to a broad range of psychosocial variables predictive of morbidity and mortality. This includes (1) a lack of social relationships and social supports; (2) personality dispositions, such as a lost sense of mastery, optimism, sense of control, and self-esteem or heightened levels of anger and hostility; and (3) chronic and acute stresses in life and work, including the stress of racism, classism, and other phenomena related to the social distribution of power and resources." ^{12(p. 1707)}

Similarly, Marmot and Wilkinson argue that the effects of poverty are deeper than material deprivation alone. Referring to the fact that black people in Costa Rica have one-fourth the real income but a life expectancy of nine more years, then,

“...the explanation for the poorer health of black people in the United States must have more to do with the psychosocial effects of relative deprivation—such as educational disadvantage, racism, gender discrimination, social and family disruption, and fear of crime—than with the direct effects of material conditions themselves. To show that social structure and relative deprivation have painful psychosocial effects is the very opposite of victim blaming. Indeed, the denial of these connections exposes the individual to blame.”^{13(p. 1235)}

The next question would be to see if what is true for mortality in general is true for diabetes risk in particular. Wamala *et al* used education as a surrogate for SES and controlled for age, menopausal status, family history of diabetes, cigarette smoking, lack of physical exercise, and alcohol consumption in looking at risk for the insulin resistance syndrome (also called the metabolic syndrome or “Syndrome X”) in middle-aged Swedish women . With these major behavioral and genetic risk factors controlled for, there was still a 2.3-fold increased risk of having the insulin resistance syndrome in the women with the least education. They argue, “...that unfavorable socioeconomic circumstances coupled with psychosocial stress may lead to a physiological defeat reaction, thereby activating the hypothalamus-pituitary-adrenocortical (HPA) axis as indicated by elevation of the major components of the metabolic syndrome, such as the waist-to-hip ratio.”^{14(p 2002)} Brunner *et al* reported on evidence from the Whitehall II Study to conclude that, “...central obesity, components of the metabolic syndrome and plasma fibrinogen are strongly and inversely associated with socioeconomic status. Our findings suggest the metabolic syndrome may contribute to the biological explanation of

social inequalities in coronary risk. Health related behaviours appear to account for little of the social patterning of metabolic syndrome prevalence.”^{15(p 1341)}

The physiological mechanisms connecting stress and trauma to risk for the insulin resistance syndrome and diabetes have been outlined. Much work has been done in this area by Bjorntorp, Rosmond and colleagues. In a recent study they state, “We suggest that the Metabolic Syndrome X is due to a discretely elevated cortisol secretion, discoverable during reactions to perceived stress in everyday life. This is based on environmental factors and expressed with different impact depending on genetic susceptibility.”^{16(p 297)} In a separate study, they conclude, “...an hypothalamic arousal syndrome, with parallel activation of the HPA axis and the central sympathetic nervous system, is responsible for development of endocrine abnormalities, insulin resistance, central obesity, dyslipidemia and hypertension, leading to frank disease, including Type 2 DM. We suggest that this syndrome is probably based on environmental pressures in genetically susceptible individuals.”^{17(p 373)}

One of the main hormonal connections between stress, the HPA axis and diabetes risk are the glucocorticoids, particularly cortisol. Corticotropin Releasing Hormone (CRH) is secreted by the hypothalamus both in a baseline circadian fashion and in response to stress. CRH stimulates the pituitary to secrete ACTH, which, in turn, stimulates the adrenal release of cortisol. Among its many physiologic effects, cortisol inhibits insulin release from the pancreatic beta cells¹⁸, stimulates hepatic gluconeogenesis, inhibits insulin action on skeletal muscle and potentiates insulin action on adipose tissue¹⁹. As such, chronic dysregulation of cortisol release secondary to chronic stress, leads to visceral obesity and the insulin resistance syndrome, thereby significantly increasing the

risk for developing type 2 diabetes. Cortisol is not the only component of the body's stress system which adversely affects insulin resistance syndrome components, the catecholamines and autonomic nervous system play large roles as well. Figure 1 is a schematic representation of the stress system published by Chrousos and Gold.²⁰

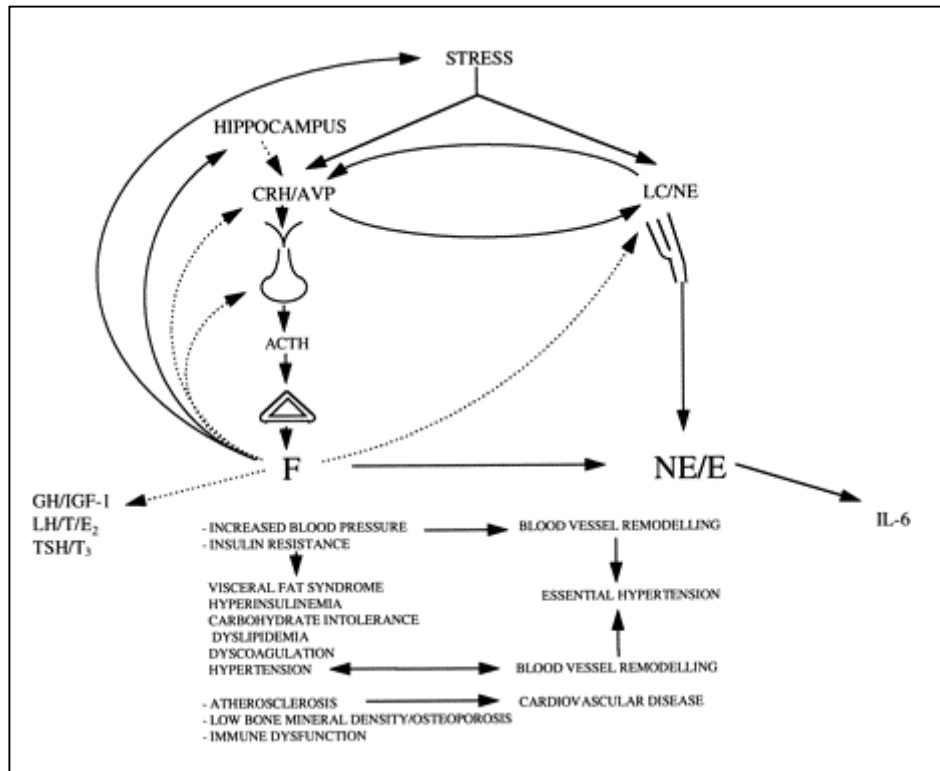


Figure 1. A schematic representation of the stress system. The CRH/AVP neurons are reciprocally connected with the noradrenergic neurons of the LC/NE system in a positive reverberatory circuit. The HPA axis is controlled by several negative feedback loops, which tend to normalize the time-integrated secretion of cortisol, yet glucocorticoids stimulate the amygdala and, hence, the fear center. Activation of the HPA axis leads to suppression of the GH/IGF-1, LH/testosterone/estradiol, and TSH/T₃ axes; activation of the sympathetic system increases IL-6 secretion. Chronic increases in cortisol, catecholamines, and IL-6 and chronic suppression of the GH/IGF-1, LH/T and TSH/T₃ axes lead to visceral obesity, hypertension, atherosclerosis, osteoporosis, and immune dysfunction and their sequelae resulting in increased morbidity and mortality. Symbols: *Solid lines* indicate stimulation; *interrupted lines* indicate inhibition. Abbreviations: HPA, hypothalamic-pituitary-adrenal; CRH, corticotropin-releasing hormone; AVP, arginine-vasopressin; LC/NE, locus ceruleus/norepinephrine system; GH, growth hormone; IGF-1, insulin-like growth factor-1; LH, luteinizing hormone; T, testosterone; TSH, thyrotropin; T₃, triiodothyronine; F, cortisol; NE, norepinephrine; E, epinephrine; IL-6, interleukin-6^{20(p 1843)}.

Further, it appears that not just current stresses affect the HPA axis. Heim and colleagues looked at the pituitary-adrenal and autonomic responses to stress in adult women with a history of sexual or physical abuse as children. They found that even

years after the abuse had ended, these women still hypersecreted stress hormones, even in response to mild stresses. “Our findings suggest that hypothalamic-pituitary-adrenal axis and autonomic nervous system hyperreactivity, presumably due to CRF hypersecretion, is a persistent consequence of childhood abuse...”^{21(p 592)} A picture is emerging of chronic stress dysregulating the release of what, in the long run, are diabetogenic and atherogenic hormones thereby significantly increasing risk for the development of cardiovascular disease and type 2 diabetes. As perceived stress rises for more people, perhaps this results in genetic thresholds for disease being reached more often.

What are the implications of these kinds of studies to Native Americans? Perhaps a new model can be developed which no longer implies that Indian people who develop diabetes were bad or lazy or that there is something defective genetically about being Indian. Both of these are destructive messages which have only further demoralized Indian people. Instead, perhaps the model can finally acknowledge what effect centuries of past and current oppression have had on the health of individuals, families, and communities. Indeed, Native Americans experience depression up to six times as often as the general population²². Depression itself is a risk factor for type 2 diabetes²³.

Psychologists Eduardo and Bonnie Duran in their book *Native American Postcolonial Psychology* argue that,

“Beginning in the late 1800’s, the U.S. government implemented policies whose effect was the systematic destruction of the Native American family system under the guise of educating Native Americans in order to assimilate them as painlessly as possible into Western society, while at the same time inflicting a wound to the

soul of Native American people that is felt in agonizing proportions to this day.”^{24(p 27)}

Further, if traumas, such as those inflicted by the boarding school system on generations of Native children,

“...are not resolved in the lifetime of the person suffering such upheaval, it is unthinkable that the person will not fall into some type of dysfunctional behavior that will then become the learning environment for their children. Once these children grow up with fear, rage, danger, and grief as the norm, it is little wonder that family problems of all types begin to emerge within the family system.”^(p 31)

Terms like “Secondary PTSD” (post-traumatic stress disorder), Historical Trauma” and “Intergenerational Grief” have been coined to refer to the far-reaching effects of trauma on the descendants of those who actually suffered it. Until the underlying issues are healed, trauma begets yet more trauma in subsequent generations . The ever-present issues of poverty and racism only make healing even more difficult and help perpetuate the cycle.

If stress and trauma, both past and present, lead to insulin resistance through neuroendocrine and behavioral/coping mechanisms, there is little wonder there is a diabetes epidemic surging through Indian country. After her work among the Navajo, Kathleen Huttlinger concluded, “diabetes needs to be considered a manifestation of intergenerational PTSD.”^{25(p 13)}

Figure 2 is an attempt toward defining an expanded model of diabetes risk in Native Americans. Please note, however, that by just changing some of the historical specifics, the same model could apply to African Americans and Hispanics, the other two minority

groups at highest risk for diabetes in this country. Is there a coincidence that family therapist Harry J. Aponte states, “The poorest in America, either through slavery (African Americans), conquest (Native Americans), or colonization (Puerto Ricans), have lost much of their original cultures. These cultures once told them who they were and gave them values that helped structure their families and communities. With these cultures there also came purpose...”^{26(p 3)}

As we enter the 21st century, however, obesity and diabetes are no longer the province of low income and minority communities alone, they are becoming plagues for white Americans as well. What is it in our increasingly stressed, overworked, disconnected society that causes our own behaviors, genetics and physiology to work against us? In a sense, the disease components of the insulin resistance syndrome are the natural end points of life lived with chronic stress. Perhaps our model of diabetes needs to expand to include the life experiences of our patients and not just their behaviors.

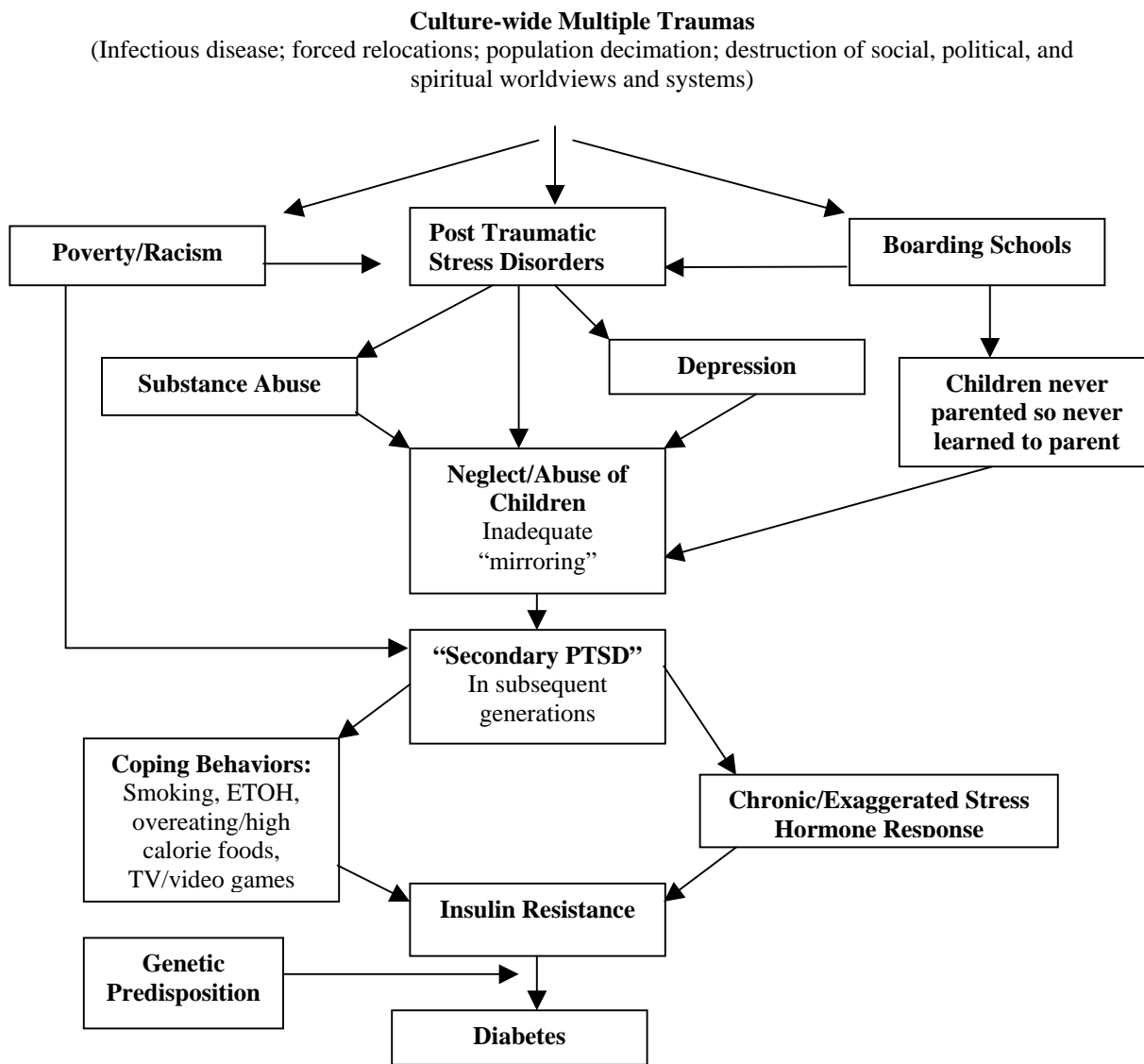


Figure 2. Proposed model linking culture-wide and individual traumatic experiences with diabetes risk.

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