Poverty versus genes: the social context of Type 2 diabetes

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Together with its 'twin sister', childhood obesity, Type 2 diabetes is spreading among young people around the world. This constitutes a serious public health problem; by their 30s, generations of young people will have been living with Type 2 diabetes for 15 to 20 or more years. The condition already accounts for up to half of the new cases of diabetes in some child populations.

However, while it is true that both obesity and Type 2 diabetes are affecting children worldwide, neither condition is affecting all children equally. Apparently, those children and adults at high risk for Type 2 diabetes come not only from non-white racial or ethnic backgrounds but more importantly, also from poor ones.1 In this article, Claudia Chaufan investigates the impact of socio-economic factors on Type 2 diabetes in young people.

In simple terms,Type 2 diabetes and the disparities in its distribution are usually explained by two main paradigms: genes and lifestyle.Type 2 diabetes in adults and young people is seen as resulting from a genetic predisposition triggered by lifestyle factors. However, the search for diabetes-specific genes has provided inconclusive evidence for such a candidate. In the USA, as genes labelled as markers of risk are identified successively in Native Americans, Mexican Americans and African Americans, the candidates multiply and the genetic picture of Type 2 diabetes looks increasingly puzzling.

The alleged connection between genes and ethnicity becomes truly problematic in cases such as Appalachia, USA where rates of Type 2 diabetes have been steadily increasing over the past 20 years among the working poor; most of the people affected there are white. On the other hand, research has shown beyond any reasonable doubt that lifestyle and Type 2 diabetes are closely linked, and that risk for Type 2 diabetes can be reduced dramatically with lifestyle modifications, irrespective of heredity.2 It would be reasonable to conclude that the best strategy to tackle Type 2 diabetes is through massive educational campaigns to develop healthy habits at an early age, to raise awareness of the dangers of the condition, and to advocate early diagnosis and treatment. Unfortunately, this is not entirely the case. Medical research has suggested that high risk for Type 2 diabetes is gestated in the womb, long before a person develops a lifestyle – healthy or unhealthy.3

High risk for Type 2 diabetes may be gestated in the womb, long before a person develops a lifestyle.

The thrifty genotype theory

The Pima Native Americans in Arizona, USA have perplexed the medical community for decades with their staggering rates of Type 2 diabetes both in adults and young people.While this trend could well be attributed to the westernization of lifestyle, it is believed that a so-called 'thrifty genotype' could explain why lifestyle had this remarkable effect on the Pima community and not on other groups.

The thrifty genotype theory contends that a predisposition to diabetes and obesity might have developed over time as a genetic response to conditions of 'feast or famine'.Today, in an environment of easily accessible oversupply of food rich in fats and sugar, this gene no longer serves an evolutionary, survival-enhancing purpose.According to the theory, this is largely responsible for the staggering rates of diabetes among certain racial and ethnic communities.

Thrifty genes aside, the historical evidence for feast or famine conditions is inconclusive. In contrast, the history is well documented of the starvation suffered by the Pima people at the turn

of the 20th century, followed by a shift to westernized lifestyles in the reservations.3 Fetal malnutrition substantially increases the risk of coronary artery disease and **impaired glucose tolerance** later in life.4 Fetal malnutrition tends to co-exist with poorly monitored pregnancies – all too common among people who are poor. Furthermore, poorly controlled gestational diabetes, also sadly frequent among under-resourced communities, increases the risk for Type 2 diabetes in children later in life.5 This congenital risk is aggravated by the calorie-dense, sedentary features of today's disadvantaged social environments.

Whether or not this is combined with the yet-to-be-discovered diabetesprone gene, the results are dramatic.

Social factors unrecognized

Interestingly, social deprivation in an environment of relative nutritional abundance, a characteristic of contemporary poverty, rarely figures in the literature among the risk factors for Type 2 diabetes. In November 2003, a search in PubMed (a searchable database of publications in the biomedical literature) with the terms 'Type 2 diabetes' and 'First Nation Canadians' combined with 'poverty' (or functional equivalents) retrieved zero articles.

*A person has **impaired glucose tolerance** (IGT) when their blood glucose levels are higher than normal, but below the level of a person with diabetes. People with IGT are at increased risk of developing Type 2 diabetes.*

The same terms combined with 'lifestyle' retrieved two articles – a small number given the role of lifestyle in the risk of Type 2 diabetes.A search with 'genes' retrieved 14 – a relatively large number given the inconsistent evidence of a genetic variation which may explain risk disparities among racial groups. Another notable absence in the literature searched was that of the variety of social factors, including aggregate measures of social wellbeing, as well as income, education and occupation. Although these have been shown to account for a variety of medical conditions, such considerations in Type 2 diabetes were marginal.6

Social deprivation in an environment of relative nutritional abundance rarely figures among the risk factors for Type 2 diabetes.

It is not a deprived social environment rather than unhealthy behaviours which explains the disparities in Type 2 diabetes; both of these can and often do co-exist. It may well be that a person who is poor is more likely to eat cheaply (in fast-food outlets) and have other unhealthy habits, such as smoking due to unmanaged stress or drinking due to depression provoked by debt or unemployment.

These factors increase the difficulty for a person in these circumstances to encourage healthy lifestyles in their children. The decisions about what, as a society, we do about Type 2 diabetes, will depend on where we focus our attention in the chain leading to Type 2 diabetes.

Treating the symptoms, missing the condition

There may not be much difference between racial and ethnic groups regarding the factors which actually put them at risk. For instance, the social and financial constraints against healthy, diabetes-free lifestyles among a Mexican American community in Northern California point to a more fundamental commonality between these people and the working poor in Appalachia regarding Type 2 diabetes than any particular fact about their genes or individual behaviours.7

Together with fetal malnutrition and poorly controlled gestational diabetes, these social and financial factors explain why Type 2 diabetes is not an equal-opportunity condition. In the production and reproduction of racial and ethnic disparities in Type 2 diabetes, the starting point is the same one regardless of the genetics: social deprivation.

Whatever the particular genetic mechanisms leading to Type 2 diabetes, social deprivation is driving the disparities in the distribution of risk for the condition. If we are to help conquer the condition and not merely treat its symptoms, health professionals need to assume roles as advocates for better overall social conditions. In this article, my thoughts about a social causation for the condition make reference only to Type 2 diabetes. Unlike Type 2 diabetes, Type 1 diabetes is not distributed in any systematic way along social categories. Its onset, to the best of my knowledge, has little, if anything, to do with social deprivation. Nevertheless, once diabetes has developed, my suggestions about the impact of social factors on the development of complications are relevant to all forms of the condition.

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