Sweet Blood:
Can Rapid Economic Growth Trigger Type 2 Diabetes?

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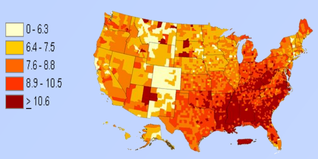
Introduction

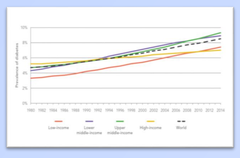
Type 2 diabetes is a metabolic health condition in which the pancreas makes insufficient insulin, a hormone that regulates blood sugar, or the body becomes resistant such that cells cannot properly utilize glucose. As a result, excess glucose circulates in the blood stream, which over time damages many structures including blood vessels and nerves.

In recent decades the prevalence of type 2 diabetes has surged in the southern United States and in many middle-income countries, and in particular among economies with a long history of poverty followed by rapid economic growth.



Age-Adjusted Estimates of the Percentage of Adults with Diagnosed Diabetes in 2009

Source: ttp://www.cdc.gov/diabetes/data/index.html



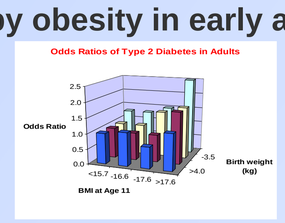
Trends in the Prevalence of Diabetes, 1980-2014, by Country Income Group

Source: WHO (2015), Table 3.4

This project explores an explanation for the connection between rapid economic growth and type 2 diabetes.

Motivating Literature

The fetal origins hypothesis (Barker, 1990) suggests that the endocrine pancreas is a form of permanent biological human capital, which is shaped *in utero* and fixed thereafter to process the diet that the net nutrition of the mother and grandmother forecast it to encounter. Generations of poverty and poor nutrition create a thrifty phenotype that will struggle to process a rich diet. Therefore unbalanced physical growth—a mismatch between development *in utero* and adult life-style will make the individual prone to diabetes. The chart below illustrates that individuals most at risk have low birth weight followed by obesity in early adolescence.



Source: (Barker, Eriksson et al. 2002)

Accepting the above, the issue is how to operationalize these ideas, i.e. to test the claim using readily-available data on per capita income and diabetes prevalence.

Procedures

My hypothesis attributes high rates of diabetes prevalence, often discovered among middle-age adults, to newly-attained, rapid economic growth during the adulthoods of these birth cohorts.

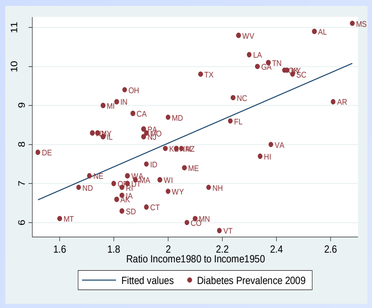
My empirical work uses aggregate data at the level of states and countries. The analysis begins with a very simple model:

$$P_i = \beta_0 + \beta_1 R_i + \beta_2 F_i + \varepsilon_i$$

where P_i represents the current age-adjusted prevalence rate for state (or country) i, R_i denotes the ratio of per capita income (or GDP) in period 2 relative to period 1, and F_i represents fixed effects. Periods 1 and 2 are designed to bracket the onset of rapid growth, which are taken as 1950 and 1980 for the U.S. and 1960 and 2000 for other countries.

Results

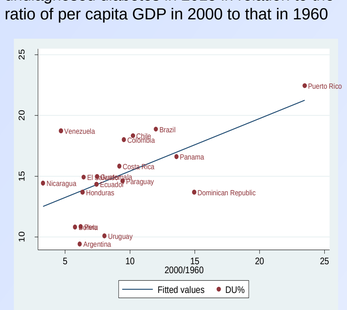
Prevalence of Type 2 Diabetes in 2009 in States of the U.S. Relation to the Ratio of per Capita Income in 1980 to that in 1950



	Coef.	Std. Err.	t	P>t	Beta coeff.
ratio 1980/1950	1.697	0.5529	3.07	0.004	0.332
Per cent black	0.0803	0.0160	5.01	0.000	0.542
constant	3.837	1.0627	3.61	0.001	
N = 50; R^2 = 0.56					

Table 1: Explaining the Prevalence of Type 2 Diabetes in 2009 by State

Age-adjusted prevalence of diagnosed and undiagnosed diabetes in 2015 in relation to the ratio of per capita GDP in 2000 to that in 1960



Vari	Coef.	S. E..	t	P>t	95% Conf.	Interval
r0060	0.214	0.063	3.39	0.001	0.089	0.339
AFR	-6.758	2.113	-3.2	0.002	-10.948	-2.568
EUR	-1.721	2.041	-0.84	0.401	-5.768	2.325
MENA	1.158	2.634	0.44	0.661	-4.064	6.380
NAC	4.526	2.681	1.69	0.094	-0.791	9.843
SACA	0.758	2.269	0.33	0.739	-3.740	5.256
SEA	4.903	3.039	1.61	0.110	-1.123	10.929
_cons	12.328	2.279	5.41	0.000	7.809	16.848
N = 113; Adj R^2 = 0.425						

Table 2: Explaining Age-Adjusted Prevalence of Diagnosed and Undiagnosed Diabetes across Countries

R summarizes a host of socioeconomic changes that triggered diabetes in the cohort of individuals born during the surge in economic growth, which are not only income itself that enables purchase of more food, but also reduced work effort associated with new technology in the factory, the field, and the household; a shift to richer diets; and more leisure. These changes promoted greater net nutrition leading to obesity, which is a major trigger for diabetes. Limited endocrine function of cohorts born on the cusp of rapid change compounded the effects. The coefficients on R are statistically significant and large in a practical sense.

In the U.S. the fixed effect is the population's share of African Americans, a group that was especially poor and who experienced a rapid rise in income from periods 1 to 2.

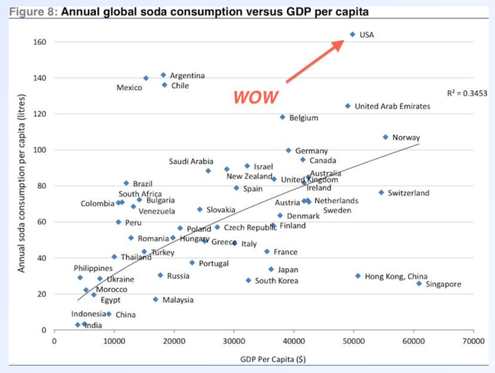
In the group of other countries, the fixed effects are dummies for region or continent. One can see that the pattern for Africa differs from that for the rest of the world in that the prevalence of diabetes was lower for a given level of GDP per capita.

Discussion

The most dramatic changes to occur in the post WWII South were agricultural mechanization, the diffusion of automobiles, and the rise of centers of industry, banking and military operations, all of which began to employ more women. The diet, however, continued to favor energy-rich foods of pork and corn and recreational exercise was rare. A reduction in work effort accompanied technological change, children's diets were increasingly unsupervised, and home-cooked meals came to be replaced by prepared foods. Obesity emerged as a health problem.

Summarizing the important changes in the rest of the world is a complex task. From the graph below, however, it seems that economic growth was accompanied by a shift to sugar consumption that may be a reasonable index of change toward a rich diet. As in the South, the effects of technological change on work and leisure plausibly accompanied income growth.

Figure 8: **Annual global soda consumption versus GDP per capita**



Conclusion

The fetal origins hypothesis indicates that the capacity of the endocrine pancreas is fixed *in utero,* and because diabetes usually appears in middle age one can link conditions of prenatal development to prevalence of the disease decades later in the same birth cohort. The former may create vulnerability and later conditions that stress endocrine function can trigger the disease.

One may criticize the empirical model on several grounds: aggregate data have well-known shortcomings, the cohort relationships are not well-matched, and GDP or income are taken as indexes of larger socioeconomic changes. It is notable, however, that despite these problems, statistically significant, powerful effects are found for income growth on the prevalence of type 2 diabetes. This discovery justifies investment of greater resources in understanding the broad socioeconomic causes of this debilitating disease.

References

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