Race and Health in the Past: Infection and Arteriosclerosis

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Abstract

Older black men experienced similar long-run declines in the prevalence of respiratory disorders and arthritis and musculoskeletal conditions as whites and greater declines in cardiovascular disorders. Using data on Union Army veterans we find very high rates of arteriosclerosis among blacks in 1910 relative to whites and to blacks today and attribute these differences to blacks' greater lifelong burden of infection. Infectious disease, especially respiratory infections at older ages, rheumatic fever, and syphilis, predicted arteriosclerosis. Additional risk factors for arteriosclerosis were being born in the 2nd relative to the 4th quarter and a low BMI.

Differences in infectious disease exposure and in susceptibility to infectious disease may explain the persistence of black-white mortality differences. Black men age 65-74 in 2003 were 1.5 times as likely as white men to die from all causes and 1.6 times as likely as white men to die from circulatory disease*. Differences in black-white mortality rates at older ages have persisted for more than a century. In the death registration states in 1900, black men's mortality rate at ages 65-74 was 1.5 times that of whites. The difference between mortality rates has remained roughly constant as well. Black men's mortality rates did not consistently fall below white men's mortality rate of 5913 per 100,000 until 1975 (1).

Older black men today not only die earlier, they also look at least ten years older than their white counterparts, epidemiologically speaking. Among black men age 50-64 in 1999-2004 three percent are sclerotic and 65 percent are hypertensive, rates not seen among white men until ages 60-74. Black men age 60-74 are more likely to have high serum cholesterol levels (a risk factor for heart disease) and lower high-density-lipoprotein cholesterol levels (a protective factor for heart disease) than whites.† Because of lack of data, previous research has not been able to determine whether differences in black and white health are as persistent as the racial differences in mortality rates.

We present the first evidence on long-run trends in black and white chronic conditions at older ages. We hypothesize that at the beginning of the twentieth century the black population's poorer nutritional status and greater life-long exposure to infectious disease relative to whites left them with higher prevalence rates for older age chronic conditions, particularly for heart disease and for arteriosclerosis, than whites.

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* Worktable 12. Death rates for 358 selected causes, by 10-year age groups, race, and sex: United States, 1999-2003 Oct 12, 2006 http://www.cdc.gov/ .

† Calculated from the 2003-2004 National Health and Nutrition Examination Survey (NHANES IV). Men were defined to be sclerotic if the ankle brachial pressure index in either the right or left leg was less than 0.7. Hypertension was defined as either taking antihypertensive medication, systolic blood pressure of at least 140 mmHg, or diastolic pressure of at least 90 mmHg. High serum total cholesterol levels are levels greater than or equal to 240mg/dl. Low levels of high-density-lipoproteins are levels less than 40mg/dl.

The black population experienced the epidemiological transition later than whites. The start of the sustained decline in black child mortality rates lagged the decline in white child mortality rates by 20 to 30 years (2) and, as DuBois (3) pointed out of Philadelphia in 1884-90, "The Negroes exceed the white death rate largely in consumption [tuberculosis], pneumonia, diseases of the urinary system, heart disease and dropsy, and in still-births; they exceed moderately in diarrheal diseases, diseases of the nervous system, malarial and typhoid fevers." We compare black and white prevalence rates at the beginning of the twentieth century using samples of Union Army veterans and investigate how these prevalence rates compare to those seen in more recent populations. We anticipate that prevalence rates for arteriosclerosis for both whites and blacks should be high relative to those observed in more recent populations.

High infectious disease rates can produce high chronic disease prevalence rates because infections 1) reduce nutritional status by decreasing food intake and increasing body loss of protein and most vitamins and minerals, 2) can cause direct organ damage, and 3) can cause inflammation that contributes to atherosclerosis (a form of arteriosclerosis), thromboses, and organ damage (4-6). Elevated blood levels of C-reactive protein (CRP), IL-6, and fibrinogen are risk factors for heart attack and stroke and these inflammatory markers are elevated during lower respiratory tract infections and during infections such as rheumatic fever, syphilis, diarrhea, malaria, and tuberculosis (6-9). Several studies have detected Chlamydia pneumonia, a bacterium that causes acute upper and lower respiratory tract infections, in atherosclerotic lesions from coronary and carotid arteries, in abdominal aneurisms, and in sclerotic and aortic valves (10). Diverse bacteria have been found in the atherosclerotic lesions of patients with coronary heart disease but not in controls. These bacteria include those involved in skin infections such as Staphylococcus species and those involved in respiratory infections such as Proteus vulgaris and Klebsiella pneumoniae and oral bacteria such as Streptococcus species (11).

Chronic infections may play a role in the initiation, progression, and destabilization of atherosclerotic plaques. Influenza vaccination is associated with a 50% reduction in the incidence of sudden cardiac death, acute myocardial infarction (AMI), and ischemic stroke, suggesting that winter peaks in stroke and AMI may arise from a destabilization of atherosclerotic plaques (12). Longitudinal studies link exposure to stress in early life (including nutritional and other insults in utero and in infancy) to the onset of chronic diseases at middle and late ages, and with waiting time to death (13-19). The exact mechanisms behind these links are still unconfirmed. For example, in some populations, older age mortality may be greater among those born in the second rather than the fourth quarter because maternal malnutrition during winter led to retarded fetal growth (18) and metabolic changes that increase susceptibility to infectious disease. Alternatively, maternal respiratory infections during the winter months could have led to placental inflammation that impaired fetal growth (6).

We hypothesize that, among Union Army veterans, observations of recent respiratory infections, infectious disease earlier in life, and month of birth predict arteriosclerosis at older ages. We also hypothesize that Body Mass Index (BMI) is negatively correlated with older age arteriosclerosis because repeated acute or chronic infectious disease will lead to poor current net nutrition.

Previous evidence on health outcomes for historical populations has found a relationship between childhood mortality rates and later cohort mortality, including those

from arteriosclerotic heart disease (5, 16). We present the first evidence on the relationship between the actual incidence of infectious disease and arteriosclerosis in a past population.

Data and Methods

Our findings are based upon two longitudinal datasets created from the military and pension records of Union Army veterans (available at http://www.cpe.uchicago.edu). The white sample is based upon the military service records of almost 35,000 men and the black sample is based upon the military service records of almost 6,000 men. The military service records provide information on events such as wartime injury and illness, age, and place of enlistment.

Both white and black veterans were eligible for a pension for war-related injuries, but because relatively few blacks were in fighting units, most African-American veterans could not claim a war-related injury. Prior to 1890, the Pension Bureau admitted 81% of white applicants onto the pension rolls but only 44% black applicants. However, beginning in 1890 pensions were paid for any disability, regardless of its relation to the war, and the Pension Bureau began to consider age 65 or older a disability in its own right unless men were "unusually vigorous." By 1907 old age (defined as age 62+) was officially recognized by Congress as a disability. Among all men who identified themselves as Union veterans in the 1910 census, we found 86 percent of the white veterans and 79 percent of the black veterans in the pension records.[‡] Among pension applicants under the 1890 law, 74% of blacks applying between 1890 and 1899 had their pension application approved by 1899 compared to 82% of whites. Pension awards under the 1890 law ranged from $6 to $12 per month and the mean pension award was 80¢ higher for whites than blacks.

Pension applications included detailed medical examinations both for men whose pension application or bid for a pension increase was rejected and for men whose bid for a pension increase was accepted. A surgeons' exam is available for 93% of all men who had a pension in 1900. Men for whom a surgeons' exam is missing tended to be men who entered at a late age and received a pension on the basis of age. Although we assume that these men did not have the specific chronic conditions that we examine, our estimates of prevalence rates are virtually the same when we omit these men from the sample.

The white Union Army sample is representative of the general Northern population before the war in terms of wealth and circa 1900 in terms of mortality experience [21]. The black sample draws disproportionately from Northern states. Twenty-six percent of all recruits came from the free states, 22 percent from the border states and the District of Columbia, and 50 percent from the Confederacy. Three-quarters of all Colored Troops were former slaves. In the Confederacy there were active recruitment efforts whenever an area was liberated by the Union Army. Thus, states that were occupied before the end of the war provided the most men.

[‡] Soldiers who survived the war were less likely to have a pension if they were deserters (and therefore ineligible), if they had never been injured in the war, if they had never been promoted, and if they were from a regiment that saw little fighting. Black soldiers were also less likely to have a pension if they had been born in the Confederacy, if they were free men at enlistment, and if they were dark-skinned.

The records of the examining surgeons include detailed descriptions of broad disease groups (e.g. cardiovascular or respiratory). For example, for the heart physicians described pulse rate characteristics; whether a murmur was present and its timing, type, and location and which valves were involved; whether the murmur was accompanied by a thrill; whether there was enlargement, edema, cyanosis, dyspnea, arteriosclerosis, or impaired circulation. Respiratory examinations included reports of respiratory sounds such as murmurs, rales, crepitation, vocal fremitus, and ronchae and reports of decreased breath sounds. Descriptions of rheumatism included where the rheumatism was located and whether pain, tenderness, swelling, or crepitation was associated with the joint. Disease rates are based upon an examining surgeon ever having noted a specific condition, symptom, or sign. Prevalence rates for 1910 may be underestimated because men who qualified for a pension on the basis of age, as many did in 1910, have fewer surgeons' exams than their counterparts who qualified on the basis of health.

The examining physicians often provided summary disability ratings for each broad disease group that were used by Pension Bureau to determine the size of the pension award. Whites had a significantly higher probability of being rated across almost all disease categories, sometimes 2-4 times as high. The exceptions to this were cardiovascular disease, arthritis and other musculoskeletal conditions, and injuries. In rating veterans, examining surgeons were more likely to designate whites than blacks as disabled. For example, by 1900, 4.5% of black examinees were blind in at least one eye, compared to 4.0% of white examinees, but among these blind veterans the examining surgeons rated only 44% of blacks as disabled compare to 88% of whites. Given the systematic downward bias in designating blacks as disabled relative to whites, it is highly likely that examining physicians under-reported the chronic conditions of blacks relative to whites.

We compare the Union Army data with random samples of the non-institutionalized population drawn from the 1971-75, 1976-80, 1988-94, and 1999-2004 National Health and Nutritional Examination Surveys (NHANES I, II, III, and IV). These surveys include medical exams, which, while not strictly comparable across all years, yield descriptions and diagnoses that can be compared with those of physicians working under contract from the Pension Bureau. The symptoms, signs, and conditions that we examine did not require any diagnostic equipment that was unavailable to late nineteenth century physicians. These symptoms, signs, and conditions are arteriosclerosis (detected by feeling whether the arteries had hardened); valvular heart disease (murmurs involving the mitral and aortal valves); congestive heart failure (concurrent presence of edema, cyanosis, and dyspnea); irregular pulse; adventitious sounds (murmurs, rales, crepitation, vocal fremitus, and ronchae); decreased breath; joint problems; and, back problems.

We compare prevalence rates by race for Union Army veterans aged 50-64 in 1900 and 60-74 in 1910 with point prevalence rates for men in the same age groups from more recent health surveys. By examining men in 1900 and in 1910 we are maximizing the number of men in those age groups. We use population weights for men in recent health surveys and weight our prevalence rates for black veterans by the geographic distribution of the black population in 1900 in the four census regions. The Union Army samples are restricted to men on the pension rolls by 1900 and 1910.

We investigate the determinants of the probability of developing arteriosclerosis between 1900 and 1910 using a series of probit models. The typical probit equation that we estimate is

$$\Pr(A_{1910} = 1 | A_{1900} = 0) = \Pr(\varepsilon < X'\beta) = \Phi(X'\beta)$$

where A_t is equal to one if the veteran had arteriosclerosis at time t (=1900,1910); Φ() is a standard normal cumulative distribution function; and X is a vector of control variables. The effect of a unit change in one of the independent variables on the probability of having arteriosclerosis is given by the partial derivative of the probit function P with respect to that independent variable. Our control variables include chronic conditions, symptoms and signs in 1900 in our first specification; chronic conditions, symptoms, and signs in 1910 in our second specification; war-time illnesses in our third specification; and war-time illnesses and quarter of birth in our fourth specification. All regressions control for age in 1910 and occupation circa 1900. The first three regressions control for race. Our fourth specification is restricted to white veterans because month of birth is unavailable for blacks.

Results

Trends

Tables 1 and 2 give prevalence rates by race for Union Army veterans aged 50-64 in 1900 and for veterans aged 60-74 10 years later and point prevalence rates for men in the same age groups in recent health surveys. At ages 50-64 black veterans had higher rates of joint problems, irregular pulse, and murmurs than whites but for the most part, resembled their white counterparts. At ages 60-74, black rates of arteriosclerosis and congestive heart were much higher than those of whites. Sixteen percent of blacks had arteriosclerosis compared to 9 percent of whites and forty-seven percent of blacks had congestive heart failure compared to 9 percent of whites.

Prevalence rates for both blacks and whites were much higher than in recent surveys. Prevalence rates for the average combined category of decreased breath and adventitious sounds fell by .2% per year for both whites and blacks between 1900 and 1910 and 1988-94. For men age 50-64 prevalence rates for musculoskeletal conditions fell by .5% per year from 1910 to 1988-94 for black men and by .4% per year for white men. Within the same age group prevalence rates for the combined category of arteriosclerosis, valvular heart disease, and congestive heart failure fell by .4% from 1910 to 1976-80 for black men and by .2% per year for white men.

How reliable are these estimates of declines in prevalence rates? Physicians diagnosed arteriosclerosis by feeling a literal hardening of the arteries. A medical text of the era notes, “The increased arterial tension, thickening of the temporal, radial, bronchial, and femoral arteries, which may be recognized by the hard, cordlike feel; the hypertrophy of the left ventricle, as shown by dullness to the left and downwards; and the accentuation of the second aortic sound, --- make a group of symptoms that can hardly be mistaken for those of any other lesion” (21). The examining surgeons’ use of hard arteries as a detection criterion provides evidence of peripheral arteriosclerosis which may be evidence of atherosclerosis (cholesterol and fatty plaques in the blood) and suggests other associated diseases such as diabetes mellitus or local inflammations. A more precise diagnosis of congestive heart failure might include not only edema,

cyanosis, and dyspnea, but also cardiomegaly and exclude co-existing respiratory infection and asthma. This definition reduces the prevalence rate of congestive heart failure to 6.1% among white men aged 60-74 and to 34.9% among black men of the same age.

Prevalence rates calculated from the Union Army sample are not strictly comparable to the point prevalence rates estimated from recent surveys because the pension system did not obtain exams at the same point in time. Two possible biases exist. First, because the Union Army rates are based on multiple exams, Union Army prevalence rates may be overstated relative to a case where a single exam occurred. It seems unlikely that such a large decline in prevalence rates could be explained by definitional biases alone. Second, the Union Army prevalence rate is calculated based on the health of the veteran at the last available exam before the analysis points of 1900 and 1910, in some cases several years before. This bias will understate the true prevalence. Thus, while estimated prevalence rates may not be precise indicators of true prevalence rates, there is little doubt that disease prevalence rates in the Union Army sample were much higher than they are today.

The higher prevalence rates of joint problems among blacks relative to whites are to be expected because of manual labor predominance among blacks. Circa 1900 22% of white veterans were professionals or proprietors compared to 5% of black veterans. Manual occupations both at young adult ages and at older ages were strong predictors of older age musculoskeletal conditions among white veterans (22).

The declining disease rates in Tables 1 and 2 are readily reconciled with changes in cause of death patterns observed during epidemiological transitions. Cause of death information may be a poor indicator of morbidity rates when infectious disease rates are high . Among veterans age 60-74 in 1910 46.2% of whites died of heart and cerebrovascular diseases compared to 34.8% of blacks. 16.3% of blacks died from bronchitis, pneumonia, or influenza; 3.6% died from infectious diseases; and 14.9% died from genito-urinary disease. The comparable numbers for whites were 9.7%, 1.9%, and 12.5%. Among veterans who had arteriosclerosis in 1910, 49.7% of whites died from either heart or cerebrovascular disease compared to 39.6 percent of blacks. 18.8% of blacks who had arteriosclerosis died from bronchitis, pneumonia, or influenza.

Cause of death recording may be less accurate for blacks than for whites, thereby leading to underestimates of heart disease among blacks. However, cause of death is unknown for 49% of whites and 51% of blacks, not a large difference. (Cause of death is more likely to be available if there was a surviving spouse.) Among men with known causes of death, 4% of whites have a vague cause of death (e.g. “old age”) compared to 5% of blacks.

Consequences and Causes of Arteriosclerosis Among Union Army Veterans

A diagnosis of arteriosclerosis in 1910 was the most important chronic condition that predicted ten year mortality rates among black veterans age 60-74 in 1910. In a probit regression controlling only for age, arteriosclerosis in 1910 increased blacks’ mean ten year mortality rates of .601 by .106 ($\hat{\sigma}$=.053). Valvular heart disease, congestive heart failure, and respiratory problems in 1910 were not statistically significant predictors of ten year mortality rates for blacks. In contrast, for whites, valvular heart, congestive heart failure, irregular pulse, and adventitious sounds were all statistically significant

predictors of mortality rates and arteriosclerosis was a statistically significant predictor only at the 10% level of significance.

Among men who developed arteriosclerosis between 1900 and 1910, statistical predictors of arteriosclerosis were having valvular heart disease, congestive heart failure, an irregular pulse, a sexually transmitted disease, and diabetes in 1900 (see Table 3). STDs increased the probability of arteriosclerosis by .09, diabetes by .07, CHF by .06, and valvular heart disease by .02. New cases of arteriosclerosis in 1910 were also correlated with new cases of adventitious sounds (an indicator of a respiratory infection) and of bradycardia, both of which increased the probability of arteriosclerosis by 0.03. As hypothesized, BMI, measured at ages 50-64, was a negative predictor of arteriosclerosis, with each additional BMI unit increasing the probability of arteriosclerosis by .003.

Rheumatic fever, syphilis, and unspecified fever during the war predicted arteriosclerosis (see Table 4), increasing its probability by .06, .07, and .02, respectively. Rheumatic fever during the war also predicted a higher prevalence of congestive heart failure, an irregular pulse, and valvular heart disease in 1900. Height at enlistment and size of city of residence (not shown) were not statistically significant predictors of later arteriosclerosis. Point estimates in all specifications were similar for both blacks and whites.

White men born in the 2nd quarter were more likely to develop arteriosclerosis between 1900 and 1910 than men born in the 4th quarter (see Table 4). A 2nd relative to a 4th quarter birth date increased the probability of arteriosclerosis by .02. There is no evidence that season of birth effects arise from social differences in the distribution of births, mortality selection during the war, or mortality selection effects early in life (23).

Month of birth was not a statistically significant predictor of other chronic conditions in 1910, but it was a statistically significant predictor of the probability of the surgeons' recording a non-sexually transmitted infectious disease in 1910 (the coefficient on the 2nd relative to the 4th quarter dummy was .025, $\hat{\sigma}$=.015). It was also a statistically significant predictor of the surgeons' noting valvular heart disease in 1900 (the coefficient on the 2nd relative to the 4th quarter dummy was .036, $\hat{\sigma}$=.017) and of their noting an irregular pulse in 1900 (the coefficient on the 2nd relative to the 4th quarter dummy was .041, $\hat{\sigma}$=.020). However, even controlling for infectious conditions in 1910 and valvular heart disease and irregular pulse in 1900, month of birth was still a statistically significant predictor of arteriosclerosis.

Month of birth was not a predictor of arteriosclerosis in NHANES I and II among men age 60-74, even though month of birth significantly influenced mortality rates among men in this cohort (24, 19). However, Union Army veterans born in either the 2nd or 3rd quarter were more likely to die of all causes than veterans born in the 4th quarter and stroke death rates were highest among those born in the 2nd quarter (23).

Even controlling for pre-exisiting conditions, for current conditions, and for wartime experiences, black veterans' probability of arteriosclerosis in 1910 was .08 higher than that of whites, probably in part because we cannot observe all infectious disease incidence. Tests in three United States cities in the 1920s showed that nearly 60% of adults had acquired immunity to diphtheria prior to any artificial immunization (24), but we observe only a handful of diphtheria cases. Black wartime infectious disease rates are probably understated relative to white rates, perhaps because of the difficulty the

War Department had in obtaining competent physicians for the Colored Troops (25). Although black wartime mortality rates were higher than those of whites (20% instead of 14%) and 90% of black wartime deaths were due to disease compared to half of white wartime deaths, black veterans are listed as having had statistically significantly lower wartime rates of tuberculosis, typhoid, malaria, rheumatic fever, and diarrhea. Only cholera rates were statistically significantly higher. In the pension records, the examining surgeons noted higher rates of tuberculosis and of sexually transmitted diseases among blacks than among whites, but lower rates of other infectious diseases among blacks.

Discussion

Our study shows that older black men in 1910 faced higher rates of peripheral arteriosclerosis, congestive heart failure, irregular pulse, murmurs and joint problems than whites. The long-run decline in heart conditions was greater among blacks than among whites. Because the black population experienced the epidemiological transition at a later date than the white population, the high prevalence of chronic conditions among the black population suggests that the theory of the epidemiological transition (26, 27), which emphasizes a shift from infectious to chronic disease, may be a better description of trends in the mix of chronic and acute causes of death than of trends in chronic disease rates. Infectious disease and chronic conditions may be closely linked.

We attribute blacks' higher rates of heart conditions to their higher life-long burden of infection -- blacks either had greater exposure to infection or were more susceptible to infectious disease because of poorer nutritional status. Our study shows that infectious disease at different points in the life-cycle predicts arteriosclerosis. Among both black and white men in 1910 rheumatic fever and syphilis at young adult ages and respiratory infections at older ages led to arteriosclerosis. While a large body of literature implicates upper respiratory tract infections with atherosclerosis (28), less is known about the role of rheumatic fever and of syphilis. Case studies of patients have found an association of early atherosclerosis with tertiary syphilis (29). Many infections, including syphilis, are associated with antiphospholipid antibodies, and these antibodies may contribute to the formation of atherosclerotic thrombosis (30). Elevated high sensitivity CRP levels have been found not only in patients with rheumatic fever, but also in patients with chronic rheumatic valve disease, suggesting the persistence of inflammation even after the initial infection (9, 31). Modification of low density lipoprotein (LDL) particles due to oxidation is an important step in the process of atherogenesis and antibodies against oxidized LDL have been found in many diseases, including rheumatic fever (32). However, we cannot rule out that rheumatic fever and syphilis led to peripheral arteriosclerosis but not atherosclerosis.

An additional risk factor for arteriosclerosis among men in 1910 was being born in the 2nd relative to the 4th quarter, consistent with studies implying that atherogenesis begins in utero (33). Nutritional deprivation in utero may lead to compromised immune function and therefore to higher inflammation rates. In rural Gambia, the risk of death (which comes largely from infection related causes) is ten-fold higher for adults born in the "hungry" season, even though month of birth did not affect anthropometric and haematological status at 18 months of age (34).

Poor current net nutrition (as proxied by low BMI) circa 1900 was another risk factor for developing arteriosclerosis by 1910, perhaps because men with low BMIs were suffering from infections not recorded by the examining surgeons. If much of the arteriosclerosis at the turn of the century was due to infection not to a high fat diet, a negative correlation between BMI and arteriosclerosis is expected.

A century ago, it was widely believed that "the toxins generated by certain diseases" produced arteriosclerosis. "Syphilis may head the list, and following close in train may be named alcoholism, its twin brother. Rheumatism, gout, and tuberculosis act in the same way, while typhoid fever, scarlet fever, diphtheria, influenza, and the malignant diseases may so impregnate the blood as to be considered important factors in producing the disease" (21). Although this hypothesis was later abandoned, it has re-emerged in recent years as an explanation for atherosclerosis. We do not claim that infection alone can account for atherosclerosis. Dietary patterns may influence cardiovascular disease risk through effects on inflammation and endothelial activation (35) and may explain some of the racial differences in arteriosclerosis rates observed today. Studies of mice show that chronic mild stress can induce or accelerate the development of atherosclerosis (36), suggesting that low socioeconomic status and limited legal protections may have directly worsened blacks' health outcomes. In the case of joint problems we attribute higher prevalence rates among blacks to their predominance in manual jobs.

Economic growth and investments in public health underlie the long-run decline in chronic disease prevalence rates (4) but the benefits of economic growth and of public health investments accrued later to the black than to the white population. The former slaves were illiterate and owned no property and free blacks fared little better. Contemporary observers reported a high incidence of pellagra and rickets among blacks in the postbellum era (37). As former slaves crowded into cities after the Civil War, tuberculosis and respiratory epidemics broke out and, because men who migrated often did so without their families, syphilis rates increased (38). Because blacks could ill afford self-protection measures against infectious disease, they were particularly dependent upon public health campaigns such as those against hookworm (39) and upon investments in sanitation and water filtration, investments that were extended to black areas of town later than to white areas (40).

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Table 1: Prevalence Rates for Black Men

	1900/10	1971-75	1976-80	1988-94	1999-2004
Age 50-64					
Irregular Pulse	47.3	5.0	5.9	7.7	6.1
Murmur	36.9	5.6	6.4	3.6	3.0
Valvular HD	22.0	2.8	6.4		2.1
CHF	1.1		7.0	6.3	5.3
Arteriosclerosis	2.4	2.8	0.5		2.8
Adventitious Sounds	16.5	5.1	9.0	4.2	
Decreased Breath	15.3	4.2	3.5	5.4	
Joint problems	51.4	8.5	15.4		
Back problems	47.5	22.0	20.7	27.2	
Age 60-74					
Irregular Pulse	60.4	7.7	4.3	10.1	15.5
Murmur	39.7	9.8	9.9	3.6	3.6
Valvular HD	26.0	3.4	9.3		1.5
CHF	47.4		9.7	7.8	4.5
Arteriosclerosis	16.0	4.2	2.8		7.0
Adventitious Sounds	20.3	11.6	10.4	3.9	
Decreased Breath	22.6	3.3	10.1	8.8	
Joint problems	60.7	2.5	10.6	14.0	
Back problems	55.0	32.2	24.0	26.9	

Prevalence rates in 1900 are weighted by the geographic representation of the black population in the four census regions in 1900. Sample weights are used for all other prevalence rates. All prevalence rates are physician reported with the exception of CHF in 1988-94, which is self-reported, and of murmur, valvular heart disease, and CHF in 1999-2004, which are self-reported. Unlike earlier surveys, arteriosclerosis in 1999-2004 is not based upon physician impressions but upon an ankle brachial pressure index in either the right or left leg that was less than 0.7

Table 2: Prevalence Rates for White Men

	1900/10	1971-75	1976-80	1988-94	1999-2004
Age 50-64					
Irregular Pulse	32.4	3.5	3.4	1.9	4.0
Murmur	27.9	2.6	3.7	1.9	1.5
Valvular HD	19.2	2.5	3.4		0.2
CHF	2.7		4.2	3.7	3.4
Arteriosclerosis	1.7	1.6	0.4		0.6
Adventitious Sounds	20.1	5.1	6.0	3.5	
Decreased Breath	11.9	4.2	7.4	5.0	
Joint problems	43.2	8.5	8.8		
Back problems	39.2	22.0	24.9	34.3	
Age 60-74					
Irregular Pulse	43.7	7.1	8.4	7.8	9.7
Murmur	38.7	6.6	7.6	3.5	2.3
Valvular HD	26.9	2.6	5.5		1.2
CHF	8.9		9.5	5.6	7.0
Arteriosclerosis	9.2	1.6	1.6		2.7
Adventitious Sounds	29.1	7.9	10.5	4.0	
Decreased Breath	15.4	12.9	11.6	7.3	
Joint problems	55.0	9.1	12.3	13.1	
Back problems	47.5	29.5	39.8	31.2	

See the notes to Table 1 for an explanation of the NHANES surveys.

Table 3: New Cases of Arteriosclerosis Between 1900 and 1910 and Chronic Conditions, Probit Regression

	$\partial P / \partial X$	Std. Err.		$\partial P / \partial X$	Std.Err.
Dummy=1 if in 1900			Dummy=1 if in 1910		
Valvular HD	0.023***	0.009	Valvular HD	0.051***	0.008
CHF	0.060***	0.023	CHF	0.075***	0.015
Adventitious sounds	0.008	0.009	Adventitious sounds	0.027***	0.008
Decreased breath	0.043*	0.019	Decreased breath	0.007	0.020
STD	0.085**	0.049	STD	0.089***	0.040
Other infectious	0.003	0.010	Other infectious	0.001	0.009
Diabetes	0.074***	0.031	Diabetes	0.047***	0.021
Tachycardia	0.002	0.009	Tachycardia	0.009	0.007
Bradycardia	0.006	0.019	Bradycardia	0.031**	0.016
Irregular pulse	0.021***	0.008	Irregular pulse	0.037***	0.007
BMI at age 50-64	0.003***	0.001	BMI at age 50-64	0.002**	0.001
Dummy=1 if black	0.082***	0.021	Dummy=1 if black	0.066***	0.021
Pseudo R^2	0.030			0.080	

6410 observations. The regressions also control for age in 1910 and two occupational dummies indicating if circa 1900 the veteran was 1) a farmer and 2) a professional, proprietor, or artisan. *** p < .01, ** p < .05, * p < .10. The sample is restricted to men who were on the pension rolls in 1900 and who had a surgeons' exam indicating that they did not have arteriosclerosis in 1900.

Table 4: New Cases of Arteriosclerosis Between 1900 and 1910, Wartime Illnesses and Quarter of Birth

	$\partial P / \partial X$		White Only $\partial P / \partial X$	
Dummy=1 if born				
1st quarter			0.018	0.012
2nd quarter			0.024**	0.012
3rd quarter			0.016	0.012
4th quarter				
Dummy=1 if wartime				
Respiratory infection	0.005	0.012	0.004	0.013
Rheumatic fever	0.057***	0.013	0.052***	0.014
Diarrhea	0.006	0.008	0.006	0.009
Measles	0.000	0.013	0.007	0.015
Syphilis	0.066**	0.038	0.097***	0.047
Cholera	0.056	0.031		
Smallpox	0.044	0.037	0.051	0.043
Tuberculosis	0.034	0.030	0.051	0.035
Fever	0.020**	0.009	0.022**	0.010
Typhoid	0.009	0.013	0.009	0.015
Malaria	0.000	0.019	0.008	0.022
Injury	0.006	0.007	0.006	0.008
Dummy=1 if black	0.075	0.019		
Pseudo R^2	0.022		0.021	
N	6479		5056	

*** p < .01, ** p < .05, * p < .10. See the notes to Table 3 for additional controls and sample restrictions.