

UKCPR

University of Kentucky
Center for
Poverty Research

Discussion Paper Series

DP 2009-10

ISSN: 1936-9379

Socioeconomic status, child health, and future outcomes: Lessons for Appalachia

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Preferred citation

Currie, J. (2009, Dec.). Socioeconomic status, child health, and future outcomes: Lessons for Appalachia. *University of Kentucky Center for Poverty Research Discussion Paper Series*, DP2009-10.

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Abstract

Appalachians are in poor health relative to other Americans. For example, the age-adjusted all cause mortality rate for Appalachian in 2006 was over 900 per 100,000 compared to a rate of 760 per 100,000 for those outside of Appalachia. This essay shows that health disparities start before birth—the incidence of low birth weight is 90 1,000 in rural Appalachia compared to 83 per 1,000 outside the U.S. These disparities continue through childhood and into adulthood. Moreover, although African Americans are generally in poorer health relative to white Americans, disparities between Appalachia and the rest of the U.S. are much greater for whites. These disparities are not surprising given the continued poverty of Appalachia. This paper draws on the literature to discuss the likely consequences of these disparities for the future health and well being of Appalachia's children.

While there are clearly many things about a person's background that might matter for future outcomes, research increasingly implicates health as a major factor. Given the importance of "health capital" for education and earnings (Grossman, 2000; Case, Fertig, and Paxson, 2005; Currie and Madrian, 1999; Smith, 1999), poor health in childhood may be an important mechanism for intergenerational transmission of education and economic status.

This link is disturbing in light of the poor health of Appalachians, both relative to national samples, and in relation to other residents of Appalachian states. Figure 1 shows age-adjusted mortality rates from all causes for four groups: Rural (non-metropolitan) Appalachian counties, all Appalachian counties, other regions of Appalachian states (excluding New York City and Philadelphia), and the entire U.S. Figure 1a clearly shows higher death rates in Appalachia, and particularly in rural Appalachia than in the rest of the U.S. However, death rates for African-Americans and whites are very different as shown in Figures 1b and 1c. These figures suggest that the disparity between Appalachia and other regions is mostly due to whites – mortality rates for blacks are high, but very similar between Appalachia and elsewhere.

This essay asks whether higher death rates among adults in Appalachia might possibly be due to poorer health in childhood. Lack of longitudinal data makes it difficult to test this hypothesis, but evidence presented below suggests that it is at least a plausible scenario. If it is indeed the case that current disparities are likely to have important future health effects, then the case for ameliorating these disparities becomes even stronger.

This essay first discusses the links between socioeconomic status and child health that have been documented in the literature. Section 2 documents health disparities between children

in Appalachia and elsewhere. Section 3 draws on the literature to elucidate the likely consequences of these disparities for future health and well being. Section 4 concludes.

1. Why Might Poverty in Appalachia Yield Poor Health?

In the standard model of child health production, parents are assumed to maximize an intertemporal utility function in which parental utility depends on child health (among other things) (Grossman, 2000). Parents face budget constraints that limit the time and money they can invest in child health, as well as constraints which dictate how their time and money inputs can be converted into child health.

Health inputs are valued by consumers not for their own sake, but because they affect child health, which in turn has a direct effect on parental utility. Non-market time is an input into both health production and the production of other valued non-market goods (i.e. leisure activities). This model is dynamic in the sense that the stock of child health today depends on past investments in health, and on the rate of depreciation of health capital.

This model yields several insights into why parental poverty might matter for child health. First, the budget constraint will be less binding in wealthier families, and these families will be able to purchase more or better quality material health inputs. Inputs include factors such as better quality medical care and food, as well as safer toys, housing, and neighborhoods. Set against this is the possibility that parents with a higher value of time in market work will invest less time in child health production, though to the extent that parents can purchase substitute care of adequate quality, this may not have any negative consequence.

Lower socioeconomic status is, however, not only a matter of what inputs one can afford

to buy, but also a matter of what one can do and chooses to do with the inputs one has at hand. Parents of lower SES may have different past experiences with the health care system, or health beliefs (e.g. about whether it is normal for a child to have coughing or wheezing), or different preferences. Fuchs (1992) emphasizes the rate of time preference and a sense of self-efficacy as two things that are related to socioeconomic status but might have independent effects on the way parents choose to combine inputs in order to produce child health. Cutler and Lleras-Muney (2006) also emphasize the effect of education on decision making about health behaviors. Parental education is often highlighted as something that determines how productively parents can invest in child health.

In their models of capacity formation, Cunha, Heckman and Schennach (2006) and Heckman (2007) focus on the dynamic aspects of the human capital investment model. They argue that a model in which there are “dynamic complementarities” and “self-productivity” fits the available evidence well. Dynamic complementarities imply that investments in period t are more productive when there is a high level of capability in period $t-1$. Self-productivity implies that higher levels of capacity in one period create higher levels of capacity in future periods.

Finally, children of lower SES families are likely to have worse health at birth. Studies by David Barker (for example, Barker (1998)) show that poor fetal conditions are related to a higher adult risk of disease, especially cardiovascular disease and diabetes. This relationship has come to be known as the “fetal origins hypothesis.” There is also a recent literature discussing the fetal origins of cancer (see Stiller (2004)). Thus, differences in health at birth may affect disparities in health later in life.

b) Evidence Linking Parent's SES and Child Health

Many authors have shown that children born to low SES parents have worse health at birth (see Currie, 2009 for a summary). In an important paper, Case, Lubotsky, and Paxson show that in the U.S., these gaps in health status tend to grow as children age. Currie and Stabile (2003) show that this is also true in Canada where children have access to universal health insurance, though the rate at which the relationship between income and health strengthens with age is lower in Canada.. Their results suggest that variations in the incidence of health problems are important explanations of the gap in health status between rich and poor, and that equalizing access to palliative care is not likely to eliminate these gaps.

Poor children suffer more insults to their health than richer ones. For example, Newacheck (1994), Brooks-Gunn and Duncan (1997), Newacheck and Halfon (1998), Case, Lubotsky and Paxson (2002), and Currie and Lin (2007) all show that poor children are more likely to have many chronic conditions, and that poor children are more likely to have their daily activities be limited by their conditions. Using data from the 3rd National Health and Nutrition Examination Surveys, Bhattacharya and Currie (2001) show that family income below 1.3 times the U.S. poverty line is a significant predictor of high blood cholesterol and high body mass index among adolescents even conditional on other demographic variables.

The theory sketched above suggests that persistent poverty is likely to have worse effects on health than transitory poverty, since health is a stock that will be affected by past investments, and children with low “capacities” may be less able to parlay new investments in their health capital into good outcomes. Several authors have used data from the National Longitudinal Survey of Youth to address this issue, although its health measures are rather limited. Korenman

and Miller (1997) find that children in persistently poor families have lower height-for-age. McLeod and Shanahan (1993, 1996) and Strohschein (2005) find that child mental health, and particularly aggressive behavior, may be affected more by persistent poverty than by current poverty alone.

The literature conclusively demonstrates that children of poor or less educated parents are in worse health on average than other children, even in a rich country like the United States. But this does not necessarily imply that low SES causes poor child health. It is possible, for example, that a third factor causes both poverty and poor child health. Since parents who are in poor health are likely to have lower earnings, and may have achieved less education, perhaps parents' poor health, rather than parents' low earnings or education, is causally related to poor child health. Alternatively, poor child health may reduce parental earnings. The limited evidence on this point does not suggest large effects, however (see, Powers, 2001; Wolfe and Hill, 1994).

It is important to identify causal effects because if parental SES does not affect child health (at least when SES is above some minimum level) then interventions to increase parental SES will not necessarily improve child health. However, the literature attempting to identify causal impacts of parental SES on child health in a developed country context is small. It is difficult to find interventions that affect parental SES, but that are not also likely to have a direct effect on children's health.

Currie and Moretti (2003) use the great expansion in the number of colleges that occurred in the U.S. in the 1960s and 1970s as an instrument for college going among American women. They show that higher rates of college attendance improve infant health. College attendance also increases the probability that a new mother is married, reduces parity, increases use of prenatal

care, and substantially reduces maternal smoking, suggesting that these may be important pathways for the ultimate effect on health.

Carneiro, Meghir and Parey (2007) examine the effect of maternal education using data from the NLSY. They instrument maternal education using local labor market conditions, and the presence of a four year college and college tuition at age 17 in the county where the mother resided when she was 14 years old. They find strong effects on measures of cognitive outcomes, and even stronger medium term effects on a measure of behavior problems, which might be regarded as a proxy for or correlate of mental health conditions. They also find strong effects of maternal education on the home environment, measured using variables such as whether the child is read to, and whether the child has special lessons. They conclude that the effects of increases in maternal education are large relative to the effects of other interventions designed to affect child outcomes.

A few papers attempt to look at the effects of exogenous changes in household income. The problem here is to find exogenous sources of variation in household income that do not also affect other conditions in the household. For example, some welfare-to-work experiments increased household income, but these interventions also encouraged maternal employment, so they cannot be regarded as purely income interventions. Smolensky and Gootman (2003) summarize the welfare-to-work literature and find that over all, these interventions had surprisingly little impact on children, positive or negative.

There is some evidence that changes in family circumstances can affect childhood mental health conditions. In particular, the Moving to Opportunity experiment, which randomly assigned some public housing residents to move to low poverty neighborhoods while control

households stayed in public housing projects, found improvements in the mental health of girls. Specifically, the experimental group experienced reductions in generalized anxiety disorders and psychological distress. Curiously, there was no such positive effect for boys (Orr et al., 2003).

Costello et al. (2003) discuss the Great Smoky Mountains Study, a natural experiment involving the opening of a casino on an Indian reservation in western North Carolina. As a result of the opening, every family on the reservation received a cash transfer, but not every family was raised out of poverty by this transfer. Costello et al. compare families who were and were not raised from poverty, before and after the casino opening.

They find that before the opening, the poor children on the reservation had higher levels of psychiatric symptoms. After the opening, children in families who were raised above poverty had lower numbers of conduct and oppositional disorders (though there was no effect on anxiety and depression). However, these families were also less likely to be single headed, and reported that they were better able to supervise their children after the change, suggesting that there may have been other factors in addition to income at work.

Several papers attempt to control for unobserved family background characteristics that might be associated with both low income and low birth weight by estimating models with sibling fixed effects. Conley and Bennett (2000, 2001) use data from the Panel Study of Income Dynamics. They find that income during pregnancy has no effect on the risk of low birth weight when the mother's birth weight is controlled, or when family fixed effects are included in the model. However, they suggest that if the mother was low birth weight, then income at the time of the birth has a significant impact on the probability that the child is low birth weight in models that include mother fixed effects. Similarly, Johnson and Schoeni (2007) estimate sibling fixed

effects models estimated using the PSID and show that increases in income increase birth weight by much more if the mother was low birth weight herself.

Currie and Moretti (2007) examine intergenerational transmission of low birth weight using a much larger data set based on birth records from California. They define the mother's birth socioeconomic status by examining income in the zip code of the hospital where the mother was born. They find that mothers who were born in poor areas were both more likely to be low birth weight, and about 6 percent more likely to eventually deliver a low birth weight baby themselves, even in models comparing mothers who were sister (i.e. models including grandmother fixed effects).

In summary, there is a strong and exceedingly robust correlation between various measures of parental background and child health, and some evidence that suggests that the relationship is causal. Hence, given persistent poverty in Appalachia, it would not be surprising to see poor infant and child health.

2. Child Health in Appalachia

It is remarkably difficult to document differences in child health between Appalachia and other regions given publicly available health data, because most health data sets do not provide data on counties. Many data sets, such as the National Health Interview Survey and the National Health and Nutrition Examination Surveys, do not even provide information about state of residence in their public use files. Other data files, such as hospital discharge records or Medicaid claims data are both difficult to access and maintained at the state level, which means that there is no uniform national data base to draw on.

In this section, we will draw on the vital statistics natality and mortality data sets in order to compare Appalachian counties to non-Appalachian counties in the same states, as well as to other areas of the U.S. Vital statistics data have the advantage of being comprehensive, since they include virtually all U.S. births and deaths. In addition to vital events, they record a great deal of information about parental background as well as parent and child health conditions. Some further information about the data sources for this paper appears in Appendix 1.

Figures 2 and 3 present data on infant mortality rates, one of the most commonly used summary measures of child health, and mortality rates for all children 0 to 19, for all races and for whites and blacks separately. The figures show a pattern very similar to that of Figure 1. Although mortality rates are falling over the period shown here, they are higher in Appalachia, and particularly in rural Appalachia than elsewhere for whites. It is remarkable that in non-Appalachian regions of Appalachian states, white infant and child mortality rates are almost identical to national rates, so the disparity between Appalachia and elsewhere is not due to anything that is happening at the state level. For blacks, the rates are somewhat noisy, but there is little evidence that Appalachian rates exceed those for the country as a whole. However, rates for blacks are so much higher than those for whites that adding the two groups together (as in Figure 2a) obscures the very clear pattern for whites.

Figure 4 shows the incidence of low birth weight, the most widely used summary measure of infant health at birth. The figure suggests that the seeds of poor health are sown before children are born. Among whites, rates are persistently higher in Appalachia than elsewhere and the rates seem to be not only increasing, but diverging somewhat from the U.S. average over time. Among blacks, rates of low birth weight are quite similar in Appalachia and

the rest of the United States at the beginning of the sample period, but begin to diverge in 1995.

This pattern does not auger well for the future health of black Appalachians. Note that one might expect some mechanical correlation between falling infant death rates and rising rates of low birth weight if it is primarily low birth weight infants who are saved by improvements in medical care.

However, the relative rates are such that falling death rates can explain only a small part of the increasing incidence of low birth weight.

The natality data can also be used to shed some light on the reasons for these trends. For example, is poor infant health in Appalachia due to lack of access to medical care, maternal background, or maternal behaviors? Measures of access that we can examine include whether the child was born in the same county they reside in or a different one; whether the baby was born in a hospital; whether the mother received prenatal care starting in the first trimester, and whether or not she had a Caesarian section. These outcomes are plotted in figures 5, 6, 7, 8 for all mothers, and separately for white and black mothers.

Looking at Figure 5, it is striking how many women in rural Appalachia must travel out of county to give birth, roughly 50%. Even other regions of Appalachian states have rates of over 30% compared to a U.S. rate of about 25%. However, this fraction trends up only slightly over time. Figure 6 shows that the fraction of babies born in hospital is declining in Appalachia, compared to a rising trend in the U.S. as a whole. But even in Appalachia, less than 2% of babies are born outside a hospital. Moreover for blacks, the fraction born in hospital is higher in Appalachia than elsewhere. Figure 7 shows that the fraction of women beginning prenatal care in the first trimester is generally at least as high in Appalachia as elsewhere, even in rural areas. Similarly, Figure 8 shows that rates of C-section are almost identical in Appalachia to those in the

rest of the U.S. (and rising steadily over time). One the whole, and perhaps surprisingly, the evidence regarding access to care suggests that while Appalachian women generally have poorer access to hospitals in their county of residence, delivery of prenatal care and obstetrical care may not be greatly affected. Moreover, trends in these variables are unlikely to explain increases in the incidence of low birth weight over time.

If differential access to medical care cannot explain the trends, perhaps they can be at least partially explained with reference to maternal characteristics and maternal behaviors. Figures 9 and 10 show the fraction of mothers who have less than high school, and the fraction of mothers who are less than 19 years old, respectively. Figure 9 shows that rural Appalachian mothers are considerably less likely than other mothers to have a high school education. It is striking that mothers in non-Appalachian areas of Appalachian states are actually more likely to have at least this much education than the average U.S. mother, so there is a large disparity in levels of education within these states. Figure 10 indicates that rural Appalachian mothers are also much more likely to be teenagers than mothers in other areas, though the teen birth weight is falling over time in Appalachia as in the rest of the country. Since the children of teenage high school drop out mothers would be expected to have poorer outcomes than other children, these differences in the composition of who is giving birth may explain some of the poorer birth outcomes.

Moreover, the natality data suggest that differences in maternal behaviors are likely to be responsible for much of the differences in infant outcomes between Appalachia and elsewhere. Figure 11, demonstrates that smoking is a significant problem – in rural Appalachia, 25% of mothers smoked during pregnancy in 2005, and the rate has fallen little in the past 15 years. In

contrast, in the U.S. as a whole, about 12% of mothers smoked during pregnancy in 2005, and that rate has fallen from about 20% in 1990. Thus the growing disparity in smoking rates could very well explain the widening disparity in outcomes, since smoking is a leading cause of low birth weight.

Increasing rates of maternal obesity may also be a factor. Obese women are at higher risk for many complications of pregnancy and delivery, and their infants are more likely to be high birth weight. High birth weight (often defined as birth weight over 4000 grams) has become increasingly prevalent, and an increasing concern since it has been linked to higher body weight later in life as well as metabolic disorders such as diabetes (Cedergren, 2004, Watkins et al, 2003, Sorenson et al., 1997).

There is increasing interest in the idea that high maternal weight may program the fetus for higher weight gain both in pregnancy and after birth, with the result that the obesity epidemic may tend to accelerate over time (Ludwig and Currie, 2009). Certainly, childhood obesity is an important concern with 31% of children 6 to 19 overweight or at risk of overweight (Hedley et al., 2004). While the natality data does not record maternal pre-pregnancy weight, it does record the incidence of conditions such as diabetes and chronic hypertension that are often caused by obesity and that are risk factors in pregnant women. Figure 12 shows that the incidence of chronic hypertension is higher in rural Appalachia than elsewhere, especially for whites.

In a regression analysis (not shown), measures of infant health were regressed on controls for state, month of birth, year, race, ethnicity, maternal education, and an indicator for whether the state had adopted the new version of the birth certificate. In these regressions, children in Appalachia were 7.2% more likely to be low birth weight than other U.S. children. When

controls for mother's age, birth order of the child, mother's marital status, weight gain during pregnancy and whether the mother smoked during pregnancy were added to the regression, the "Appalachia effect" dropped to 5%. Thus, these relatively crude indicators of maternal background and behaviors explained a third of the differential in low birth weight between Appalachia and elsewhere.

3. Effects of Child Health on Future Outcomes

The fetal origins hypothesis strongly suggests that conditions in utero affect not only birth weight but features such as basic metabolism, which in turn affect future health outcomes. Since adult health is strongly linked to adult economic well-being, this suggests a relationship between health in utero and future outcomes.¹

Figure 13 shows that although the incidence of cardiovascular disease, heart disease, and stroke has been declining over time, it exhibits the same pattern as in Figure 1 in that rates are higher in Appalachia, and especially rural Appalachia than elsewhere. Once again, rates are much higher for blacks than for whites but in a departure from Figure 1c, Figure 13c suggests that death rates due to these diseases are higher for blacks in Appalachia as well as for whites. Given the evidence of small disparities in black child health between Appalachian and non-Appalachian counties, this result may suggest the importance of access to medical care for these specific conditions rather than underlying disease prevalence in determining mortality from these diseases.

Figures 14 and 15 examine patterns in two other important causes of death that have been

¹Currie and Madrian (1999) summarize the evidence linking health and labor market outcomes among working age

linked to fetal and child health: Cancer and Diabetes. Trends in these diseases conform more closely to what was shown in Figure 1. That is, among whites, there are much higher death rates in Appalachia and especially in rural Appalachia than elsewhere, while for blacks, there are high overall rates, but little evidence of disparity between Appalachia and elsewhere.

Of course, adult death rates today potentially reflect child health conditions many years ago. But if the patterns for child health were similar in the past to what we see today (i.e. child health was poorer among whites in Appalachia than for whites elsewhere, which seems quite likely) then this would at least be consistent with the patterns in adult health that are observed today.

Poor fetal and child health can also have direct effects on the acquisition of skills. For example, maternal alcohol consumption can lead to permanent brain damage, as can trauma during the birth itself. Thus, we know that severe insults in utero or in early childhood can cause permanent cognitive impairments. The question is really how sensitive these “sensitive periods” are and whether damage due to less extreme deprivation is noteworthy or widespread.

Among older children, some commentators have focused on school absences as a mechanism for health to affect education (Grossman and Kaestner, 1997). However the mean number of days absent is generally quite small for both poor and non-poor children. Hence, if poor health among school aged children has an effect on the acquisition of skills, it is more likely to come through impairing children’s ability to learn while they are in school. Conditions such as anemia and lead poisoning would have this effect. Conditions such as dental caries and ear infections are much more common so they might have a greater overall impact.

adults. The relationship between SES and outcomes may be weaker for older adults.

The most compelling examinations of the fetal origins hypothesis look for sharp exogenous shocks in fetal health that are caused by conditions outside the control of the mother. For example, Almond and Mazumder (2005) use data from the U.S. Survey of Income and Program Participation to follow cohorts who were affected by the influenza epidemic of 1918. The epidemic struck suddenly in the fall of 1918 and was largely over by January 1919. Approximately a third of women of child bearing age were infected. They show that compared to cohorts in utero either just before or just after the epidemic, the affected cohorts were more likely to suffer from diabetes, stroke, activity limitations, cancer, hypertension, and heart problems and reported poorer general health status as adults.

Almond (2006) examines the effects of the influenza epidemic on the education and labor market outcomes of people affected by the disease in utero. He finds that children of infected mothers were 15 percent less likely to graduate from high school, and that the wages of affected men were lowered by 5 to 9 percent. Moreover, affected individuals were more likely to be poor and to be receiving transfer payments (in part because they were more likely to be too disabled to work). Thus, this natural experiment provides compelling evidence that negative shocks to health in utero can have very significant effects on future economic outcomes.

An alternative way to demonstrate the importance of fetal conditions is to look at the long term effects of low birth weight using sibling/twin comparisons. Several recent studies use large samples drawn from vital statistics records in Scotland, Norway, Canada, and the U.S. All show a link between low birth weight and lower educational attainment. For example, Black, Devereux, and Salvanes (2005) find using twin fixed effects that a 10 percent increase in birth

weight leads to a one percentage point increase in the probability of graduating from high school and a one percent increase in earnings.

In addition to examining the effects of mother's SES on birth weight, Currie and Moretti (2007) use the California birth certificate data to examine the long term effect of maternal low birth weight. When they compare mothers who are sisters they find that the sister who was low birth weight is three percent more likely to live in a poor area at the time she delivers her own child, and three percent less likely to be married when she gives birth. The low birth weight sister also has about a tenth of a year less education on average.

Johnson and Schoeni (2007) examine the long-term effects of low birth weight using data from the PSID and sibling fixed effects models. They find that low birth weight is strongly related to poorer adult health and lowers adult annual earnings by 17.5 percent. Siblings who are low birth weight are less likely to have any earnings (by 4.8 percentage points). A relatively small part of this reduction in earnings is mediated by lower educational attainment—low birth weight siblings are 4.8 percentage points more likely to drop out of school, and completed education is a tenth of a year lower.

Almond and Chay (2005) examine the effect of a mother's health at birth (and in early childhood) on the health of her children using a different identification strategy based on the comparison of cohorts. They build on previous work showing that the Civil Rights movement had a large effect on the health of black infants in certain southern states, especially Mississippi (one of the Appalachian states), due to desegregation of hospitals and increased access to medical care (Almond, Chay, and Greenstone, forthcoming). For example, there was a large decline in deaths due to infectious disease and diarrhea in these cohorts.

Because birth records include the mother's state of birth, it is possible to identify black women who benefited from these changes (the 1967 to 1969 cohorts), and to compare the outcomes of their infants to the outcomes of infants born to black women in the 1961-1963 birth cohorts. The birth outcomes of white women in the same cohorts are examined as a control. Almond and Chay conclude that the infants of black women who had healthier infancies as a result of the Civil Rights movement show large gains in birth weight relative to the infants of black women born just a few years earlier, and that these gains are largest for women from Mississippi – the most affected state. The estimates indicate that the black-white gap in the incidence of very low birth weight was 40 percent lower in children of mothers from the 1967-1969 cohort compared to the earlier cohort.

Although this is a historical study, the results may be particularly relevant for Appalachia today given the evidence discussed above that Appalachian mothers continue to have poorer access to health care than other mothers in their states. Moreover, if county of birth could be identified in the mortality records, and if county-level health measures could be obtained dating back to decedent's year of birth, then it might be possible to do similar analyses using the Vital Statistics Mortality data, focusing on the long-term effects of early health indicators.

In summary, there is a great deal of evidence that fetal health affects future child outcomes. It is notable that the effects on education are smaller than those on future earnings. It is also remarkable that the evidence is so consistent across countries and time periods, that the effects appear to be roughly linear, and that the inclusion of maternal fixed effects (i.e. sibling comparisons) produce estimates that are roughly similar to OLS estimates. Hence, if fetal health was poorer in rural Appalachia than elsewhere over the past 50 years (as seems likely), it is not

surprising to observe that many Appalachians remain poor and subject to higher death rates years later.

b) Child Health Problems

Relative to the many recent studies examining health at birth, there are few studies examining other health measures, or measures for older children. Smith (2009) investigates the relationship between child health and future outcomes using data from the 1999 Panel Study of Income Dynamics. The adult children of the Panel Study of Income Dynamics (PSID) respondents (who were 25 to 47 years old) were asked a retrospective question about the state of their health when they were less than or equal to 16 years old: Was it excellent, very good, good, fair or poor? Smith estimates sibling fixed effects models and shows that better health in childhood is related to higher incomes, higher wealth, more weeks worked, and a higher growth rate in income in adulthood. The estimates imply that within families, a sibling who enjoyed excellent or very good health in childhood earns 24 percent more than a sibling who was not in good health. Much of this effect on earnings appears to come through effects on adult health which reduce work effort.

Currie et al. (2009) use Canadian administrative data to look at the effects of health conditions in childhood on future outcomes using sibling fixed effects models. Data on all contacts with health care providers from the public health insurance system was merged with data on future health, educational attainment, and welfare use. They examine asthma, serious injuries, an aggregate composed of all other physical health problems, and mental health problems at different stages of childhood. Their main finding is that other physical health problems in early

life predict poorer educational attainment and higher welfare use, because they predict future health problems. That is, once health problems in young adulthood are controlled, measures of physical health in childhood have little predictive value. This finding is striking in view of claims that poor health in childhood can reduce human capital accumulation by, for example, causing school absences.

They pay particular attention to injuries given that injuries rather than illnesses are the leading cause of death among children in developed countries (Bonnie et al., 1999). They present some evidence that injuries in adolescence are predictive of poor future outcomes. However, they find that early childhood injuries do not affect future outcomes unless they have lasting effects on health.

Perhaps surprisingly in view of the large literature focusing on asthma, which is the most prevalent chronic condition of childhood, Currie et al. find little evidence that asthma in childhood was predictive of either lower educational attainments or an increased probability of welfare use. Previous studies have reported an effect of asthma on school absences, the probability of having learning disabilities, and grade repetition. For example, Fowler et al. (1992) use data from the 1988 National Health Interview Survey for children from grades one to twelve, and find that the asthmatic children averaged 7.6 days absent compared to 2.5 days for well children. Nine percent of the asthmatic children had learning disabilities, compared to 5 percent of the well children, and 18 percent had repeated a grade, compared to 15 percent of the well children.

In the only study to explicitly examine school readiness, Halterman et al. (2001) examine 1058 children entering kindergarten in urban Rochester and find that asthmatic children had lower

scores on a test of school readiness skills, and that their parents were three times more likely to report that they needed extra help with learning. There were no differences, however, on tests of language, motor, and socio-emotional skills. The negative effects of asthma were concentrated among children whose asthma caused activity limitations (suggesting that it was not adequately controlled). Boys were more likely to be in this group than girls.

A difficulty with all of these studies is that since asthma is more prevalent among poor and minority children than among other children, the apparent connection between asthma and outcomes could reflect omitted third factors. The studies from the medical literature discussed above are typically correlational studies which control for few potential confounders.

In contrast to the results for physical health problems, Currie et al. report that mental health problems had an independent effect on educational attainment and welfare use, even after conditioning on future health problems. Relative to their own healthy siblings, children with an early diagnosis of ADHD or conduct disorders were 1.6 percentage points more likely to end up on welfare immediately after becoming eligible at age 18 (on a baseline of 5.5 percent). They were also 4.4 percent less likely to be in grade 12 by age 17.

c) More About the Importance of Mental Health Conditions

The MECA Study (Methodology for Epidemiology of Mental Disorders in Children and Adolescents), cited in the 1999 U.S. Surgeon General's Report on Mental Health, finds that approximately one in five children and adolescents in the U.S. exhibit some impairment from a mental or behavioral disorders, 11 percent have significant functional impairments, and 5 percent suffer extreme functional impairment (Shaffer et al., 1996; U.S. DHHS, 1999). These are very

large numbers of children. Moreover, as Currie and Madrian (1999) discuss, mental health problems are leading causes of days lost in the work place, because they strike many people of working age.

In one of the few studies to examine the issue, Costello et al. (1996) find that the rates of mental illness (including anxiety, conduct disorders, and hyperactivity among others) in Appalachian children aged 9 to 13 were quite similar to national rates. However, mental health problems may be a particular concern in Appalachia because of a lack of services for mental health conditions. One study found that while 58% of rural non-Appalachian counties in Appalachian states were underserved, 70% of rural Appalachian counties lacked mental health services (Hendryx, 2007).

Several longitudinal studies have found that children with “externalizing” mental health conditions such as Attention Deficit Hyperactivity Disorder (ADHD) or conduct disorders have significantly poorer mental health and schooling outcomes as teens or young adults (see Caspi et al., 1998; Miech et al., 1999; McLeod and Kaiser, 2004; Mannuzza and Klein, 2000). Some find that effects are particularly pronounced in children of young or less educated mothers, which would mean that Appalachian children were particularly at risk given that their mothers are more likely to fall into these categories (Nagin and Tremblay, 1999).

Currie and Stabile (2006, 2008) use sibling fixed effects models estimated on data from the National Longitudinal Survey of Youth and a similar Canadian survey. All children in these surveys were administered “screeener questions” that can be used to form a scale for ADHD. They focus on 4 to 11 year old children who had ADHD and examine outcomes 6 years later. They find that children who initially had high scores on the ADHD screener had lower cognitive

test scores than other children for math and reading (by approximately 1/3 of a standard deviation), and much higher probabilities of being in special education or having repeated a grade when they were followed up. The effects of ADHD are larger than those of other mental problems, and are quite similar in the U.S. and in Canada. Finally, the effects of ADHD are large relative to those of physical chronic conditions.

These findings suggest that mental health conditions may form an important component of the “non-cognitive skills” that have been stressed by authors such as Heckman, Stixrud, and Urzua (2006). These authors distinguish between measures of children’s cognitive skills (such as IQ scores) and measures of their noncognitive skills (such as the presence of behavior problems) and argue that the later are at least as important in explaining future success in life.

d) Toxic Exposures

Toxic exposures are an area of particular concern in Appalachia given the importance of heavy industries such as coal production in the region. Coal mining creates particulates and may lead to exposures to toxins such as arsenic, cadmium, lead, and mercury. There has been little study of the extent to which pollution due to coal mines or other heavy industries is associated with poor health.

Hendryx (2007) uses county-level data about hospitalizations from the Health Care Cost and Utilization Project to examine the way that hospitalizations for “coal sensitive” and “non coal-sensitive” conditions vary with levels coal production in West Virginia. He finds some evidence of an effect on coal-sensitive conditions, and finds that the effects are as large for

women (who are less likely than men to work in the mines than men). He interprets this as evidence that coal production may have effects on neighbors as well as on workers.

Exposures to heavy metals in childhood have serious consequences: For example, lead has been shown to decrease IQ by two to five points for each 10 to 20 microg/DL above the current standard (Pocock et al., 1994). However, the government tracks lead poisoning by looking for areas with a combination of older housing stock and low income households (since these markers are predictive of exposure to lead paint), so that surveillance is not especially precise. Lead may also have negative effects on children's mental health, making them more prone to anti-social behavior (Needleman and Gastsonis, 1991).

Before the regulation of lead, children were exposed to lead from paints, water pipes, gasoline, and canned food. Evidence from the NHANES surveys showed that 88.2 percent of children aged one to five had lead levels above 10 microg/dl in 1976 to 1980, 8.6 percent had lead levels above the threshold in 1988-1991, and only 2.2 percent had levels this high in 1999-2000. These figures imply that the number of children with unsafe lead levels declined from 13.5 million to less than one half million over this period (U.S. Centers for Disease Control, 2003).

Lead poisoning hot spots persist however, and some of them are in Appalachia (see <http://www.cdc.gov/nceh/lead/data/state.htm> for state maps showing counties with elevated lead in 2006. Note that no such data are available for Tennessee, South Carolina, or Alabama). Practices such as coal mining via mountaintop removal and failures of coal ash sludge ponds (like the one that recently occurred in Tennessee), are alleged to contaminate drinking water with heavy metals including lead (see Duhigg, 2009).

There is little research on the question of whether exposure to toxic releases at the level

that now generally occurs in the population has negative health effects. Data on possible human health effects often comes either from animal studies, or from disastrous accidental releases. Woodruff et al. (1998) run 1990 data from the U.S. Environmental Protection Agency's Toxic Release Inventory through a dispersion model and calculate that 90 percent of Census tracts have concentrations of several chemicals greater than cancer benchmarks. This suggests that American children (and others) may be at risk from toxic releases, but does not establish any direct relationship between releases and health effects.

Currie and Schneider (2009) use data from the Toxic Release Inventory and compare the effects of chemicals known to have developmental effects to those which are not known to have such effects. They also compare the effects of "fugitive air releases" (in which chemicals escape from a plant in to the surrounding environment) to those of "stack air releases" (in which chemicals are funneled up a tall smoke stack and hence disperse over a further area). They show that fugitive releases of developmental chemicals were associated with higher infant death rates. The effects were particularly large for heavy metals such as cadmium, but were also significant for commonly released volatile organic compounds such as toluene.

Vriheid (2000) looks at the question of whether residence near a hazardous waste site has health effects, and highlights some of the methodological weaknesses of existing studies. Residents of areas near hazardous waste sites are more likely to be poor and have lower levels of education than people in the remainder of the country (Currie and Neidell, 2005; Gallagher and Greenstone, 2005) so that their children's health outcomes are likely to differ even in the absence of negative health effects from exposure. Some studies control for some observable confounding factors, but there is still a possibility that there are unobservable characteristics of people who live

close to hazardous waste sites which would tend to cause bad outcomes. An additional problem is that the number of hazardous waste sites analyzed in many of the previous studies is small, so that some “results” may actually be due to sampling variability. These problems plague much of the literature on toxic effects so that it is quite difficult to measure effects of pollution on health, let alone show that there are long-term consequences of exposures.

In two important and innovative works, Chay and Greenstone (2003a,b) used changes in regulation to identify pollution’s effects on infant mortality. They argued that the 1970 and 1977 Clean Air Acts caused exogenous changes in pollution levels, and that the changes were different in different areas. These changes can be used to examine pollution’s effects on housing markets and infant mortality. They found that a $1\mu\text{g}/\text{m}^3$ reduction in total suspended particulates (a common measure of overall pollution at that time) resulted in 5-8 fewer deaths per 100,000 live births. Reyes (2005) uses variation in prenatal lead exposure caused by the Clean Air Acts on infant health outcomes, and finds that even small amounts of lead are associated with adverse outcomes.

Currie and Neidell (2005) examine the effects of air pollution on infant deaths in the 1990s. They use individual-level data and within-zip code variation in pollution over time to identify the effects of pollution. They included zip code fixed effects to account for omitted characteristics like ground water pollution and socioeconomic status, and found that reductions in two pollutants – CO and PM_{10} – in the 1990s saved over 1,000 infant lives in California. Pollution may also have many negative health effects without causing deaths. Neidell (2004) also uses within-zip code variation in pollution levels in California to provide evidence that air pollution affects child hospitalizations for asthma. Currie, Neidell, and Schmeider (2009)

conduct a similar analysis for New Jersey using individual panel data and show that in models with mother fixed effects, increases in exposure to CO are predictive of lower birth weight and prematurity.

Exposure to pesticides may also be an important concern, particularly in rural Appalachia. Rauth et al. (2006) examine a group of children who were prenatally exposed to pesticide (chlorpyrifos) and find that children in a high exposure group were five times more likely to be developmentally delayed than those in a lower exposure group. This is not an experimental study, but the results suggest that differential exposure to toxic substances might be a significant problem for poor children.

These studies show that pollution can have causal effects on child health, but there has been little investigation of whether these negative health effects have long term consequences for children's outcomes. The National Children's Study, which was authorized by the Children's Health Act of 2000, will attempt to remedy this situation by examining the effects of environmental exposures on 100,000 children who will be followed from birth to age 21 (see <http://www.nationalchildrensstudy.gov>).

e) Acute Conditions

We have seen that mothers in Appalachia must frequently travel to neighboring counties to give birth, and that there is a shortage of mental health professionals. These findings may be indicative of a general lack of access to care for acute conditions. Poor children are more likely to suffer from acute illnesses such as dental caries and ear infections than richer ones. Poor dental health in particular seems to be a significant problem in Appalachia. Crout et al. (2008)

report that by the age of 8, one-third of children have untreated dental decay in West Virginia, and one-third of West Virginians under age 35 have lost at least six permanent teeth.

Ear infections (otitis media) affect most young children at one time or another and are the most common reason children visit a doctor. They can be extremely painful, though more than 80 percent of infections resolve themselves within three days if untreated. Among children who have had acute otitis media, almost half have persistent effusion after one month, a condition that can cause hearing loss. Researchers estimate that at any given time roughly 5 percent of two- to four-year-old children have hearing loss because of middle ear effusion lasting three months or longer (O'Neill, 2003). Hearing loss can delay language development.

The prevalence of acute conditions is not well monitored in national data, so that it is difficult to ascertain how prevalencies of conditions such as ear infections compare between Appalachia and the rest of the country.

4. Conclusions

Health is a multi-dimensional concept, and ideally we would like to account for the effects of all of the health insults suffered by a child and the possible interactions between them. As we have seen above, long-term effects of low birth weight on education are statistically significant and found in many different settings, but are relatively small (on the order of a tenth of a year of education, on average). However, this is just one of a number of health insults that a child may be exposed to. For many important categories of health insults, such as injuries and environmental exposures, we do not have accurate evidence regarding the likely long-term effects, or the extent of the disparity in exposures.

Moreover, several studies suggest that health insults may have large effects on future earnings and/or employment probabilities, even if they have relatively little effect on completed education. Completed education is a crude proxy (with relatively little variation) for the possibly more subtle effects of health in childhood on cognitive functioning. Further, childhood health may affect adult outcomes by influencing non-cognitive skills, and through effects on adult health.

In contrast to the estimates for most conditions that have been routinely observed at birth or later, the fetal injuries investigated by Almond and his collaborators appear to have very large effects on future outcomes. Recall that children of U.S. mothers infected during the flu epidemic were 15% less likely to graduate from high school, while the Swedish children exposed to low-level radiation after Chernobyl were 5.6% less likely to qualify for high school. These results raise the possibility that the best way to safeguard children's health may be safeguard the health of their pregnant (or pre-pregnant) mothers. They also suggest that our standard measures of child health after birth (such as birth weight) are inadequate since they do not appear to fully capture the effects of these fetal health insults.

As discussed above, several studies have demonstrated that there are intergenerational correlations in health status, and that there may be interactions between parental health status and parental economic status in the production of child health. The fetal origins literature provides a natural explanation of why this might be. But thus far, few researchers have attempted to directly assess the extent to which intergenerational transmission of income might operate through the effects of parental income on child health, and subsequent effects of poor child health on children's adult education and income.

What are the implications of these findings for Appalachia? First, as we saw above, Appalachian adults are in relatively poor health, and are more likely to die of cardiovascular disease, stroke, cancer and diabetes than other U.S. adults. It is common to blame such disparities in outcomes on disparities in current access to medical care. However, the research summarized above suggests that they may have their roots in childhood deprivation rather than current deprivation. This observation further suggests that it will be difficult to eradicate adult health disparities in the short run.

While death rates for blacks are higher than for whites, black Appalachians do not appear to be disadvantaged relative to other U.S. blacks. These patterns may reflect the fact that all blacks were disadvantaged relative to whites 50 to 60 years ago, and that the gap between Appalachian blacks and other blacks at that time may not have been very large.

The results discussed above suggest that it is important to work to eliminate child health disparities if adult health in Appalachia is ever to be brought up to national levels. The large numbers of pregnant women who smoke or who have obesity-related conditions are of particular concern among both black and white mothers. Since both smoking and obesity are better treated via prevention than after the fact, a larger roll for public health campaigns targeting these health behaviors would seem to be warranted.

Data Appendix

The data used in this paper come from national Vital Statistics Natality and Mortality files. Public use files do not contain county identifiers, but these are available by permission from the National Center for Health Statistics.

Geographic Category Definitions

1. *Appalachia* - any county in Appalachia. The Appalachian Regional Commission (ARC), which was established in 1965 by President Johnson, defines Appalachia based on counties, and as of fall 2009, it contains 420 counties that span from southern New York to northern Mississippi. It covers all of West Virginia and parts of 12 other states. A map of the region is available at: <http://www.arc.gov/index.do?nodeId=938> .

2. *Rural Appalachia* - any county in Appalachia that was *not* a metropolitan county in 2006.

Note that while researchers commonly refer to areas that are “not metropolitan” as “rural”, the census definition is much more limited; the census defines “rural areas” as comprising open county and settlements with fewer than 2,500 residents.

Since 2003, the OMB has defined metro areas as (1) central counties with one or more urbanized areas, and (2) outlying counties that are economically tied to the core counties as measured by work commuting. Outlying counties are included if 25 percent of workers living in the county commute to the central counties, or if 25 percent of the employment in the county consists of workers coming out from the central counties—the so-called “reverse” commuting pattern. We use the 2006 classifications to avoid composition effects due to the change in definition of metro areas. More information about these definitions can be found at:

<http://www.ers.usda.gov/Briefing/Rurality/NewDefinitions/>

3. *Other Regions of Appalachian States* – an Appalachian state is a state that contains one or more counties that are in Appalachia. Other regions are the areas of these states that are *not* in Appalachia. ***New York City and Philadelphia are excluded from this group even though they are technically in Appalachian States.***

Rural Appalachia is a proper subset of Appalachia. Appalachia and Other Regions of Appalachian states are mutually exclusive categories.

Calculation of Age-Adjusted Death Rates

It is important when examining trends in death rates over time, to adjust for the age composition of the population. We have done this following the procedures outlined at:

<http://www.health.state.pa.us/hpa/stats/techassist/ageadjusted.htm> except that we use the year 2000 standard population (shown in Table A1 below), which is what the NCHS and Centers for Disease Control use for any data from 2003 or later.

This procedure involves the following steps:

1. Calculate the crude death rates for each of the year 2000 standard population age categories.
2. Multiply the population for each age group by its crude death rate to obtain the expected number of deaths for each age group.
3. Add the expected deaths up to get the total number of expected deaths, and divided the total number of expected deaths for the total year 2000 standard population (274,633,642).
4. Multiply by 100,000 to obtain the age-adjusted rate per 100,000 population.

Procedures for Using Natality Data

General Procedure for Figures

The variable means for the geographic categories were constructed from a dataset of county-year-month means. Means by geographic category were constructed by taking the means over the cells in those categories, weighting by the number of births in each cell.

Since the county-year-month means were taken over the non-missing values of variables in the cell, it is possible for means to be assigned based on a very small fraction of births in the cell. This can happen because some residents of one state that does not report a particular variable could give birth in another state that does report that variable. Therefore, if 90% or more of births in the cell having missing values for a variable, we create a missing value for that cell. Then, the means by geographic category were constructed from the non-missing cell means, weighting by the number of births in the cell.

Procedure For Prenatal Care Figures

Beginning in 2003, states began to adopt the CDC's recommended 2003 revised birth certificate. Unfortunately, the question about prenatal care differs between the 1989 birth certificate and the 2003 revised birth certificate as follows:

The wording of the prenatal care item was changed from "Month prenatal care began" to "Date of first prenatal visit." Also, the 2003 revision process resulted in a recommendation that the prenatal care information be gathered from the prenatal care or medical records, whereas the 1989 revision did not recommend a source for these data. Accordingly, prenatal care data for the two revisions are not directly comparable.

The Prenatal Care Figures are corrected for the changes in the question in the following way:

Let Y be observed first trimester prenatal care. Let X be the proportion of first trimester prenatal care under 1989 birth certificate. Let \hat{X} be the proportion of first trimester prenatal care under the 2003 revised birth certificate.

If we assume that the change in the question just results in a mean shift, we can write:

$$X_{\text{hat}} = X + \beta(\text{New_birth_certificate})$$

Then, $Y = X$ if $\text{New_birth_certificate}=0$, $Y=X_{\text{hat}}$ if $\text{New_birth_certificate}=1$

So, $Y=X + \beta(\text{New_birth_certificate})$

This implies that $X=Y-\beta(\text{New_birth_certificate})$.

We regressed the observed value of prenatal care on a constant and an interaction between the state dummy and new birth certificate. This allowed us to obtain a β_{hat_s} for each state s that changed birth certificates, effectively allowing the mean shift to differ by state. We then subtracted $\beta_{\text{hat}_s} * \text{state_dummy} * \text{New_birth_certificate}$ from Y to obtain estimates of X , and then averaged these X s over the geographic areas.

Procedure for the Regressions

Three sets of regression models were estimated, which included the variables listed below *plus* dummies for state, dummies for changes to the birth certificate described above. The regressions are weighted by the number of births in each cell and standard errors are clustered by county.

Model 1 - controls for black, Hispanic, maternal education (<HS, HS, some college, college plus), state, birth month, year and (state*after adopting new certificate). The later is included to deal with the measurement problem having to do with the revision of the birth certificate.

Model 2 - add maternal age (19-24, 25-34, 35-44, 45+), parity (1, 2, 3, 4, 5+), married, smoking, wghtgain (0-15, 16-30, 31-45, 46-60, 60+). These variables reflect maternal choices and behaviors.

Model 4 - add indicators that might have to do with access to care: prenatal care in 1st trimester, birth in a hospital, birth in county of residence.

Variable Definitions and Additional Notes

Low Birth Weight – Less than 2500 grams

White – includes Hispanic whites

Black – includes Hispanic blacks

Hispanic – Hispanic origin, includes Hispanic Whites and Hispanic Blacks

Non-Hispanic White – whites, excluding Hispanic Whites

Education – The education variable changed between the 1989 and 2003 birth certificates. In 1989, years of education were reported, while in 2003, highest completed degree is reported. We mapped 0-11 years to “Less than High School”, 12 years to “High School”, 13-15 years to “Some College”, and 16+ to “Bachelor’s Degree or More”.

Father Age Not Reported – this variable was an attempt to measure father involvement, but I think it mainly captures state reporting standards, whether the state asks or not

Father Race Unknown/Not Stated – again, an attempt to measure father involvement, but may capture state reporting standards, especially since it seems to increase as states are changing birth certificate formats

Smoking During Pregnancy – There is a change from the 1989 to the 2003 revised birth certificate; for the 1989 birth certificate, smoking is 1 if the mother reported tobacco use during pregnancy. For the 2003 birth certificate, the mother is asked how many cigarettes she smoked daily in the first trimester, how many cigarettes she smoked daily in the second trimester, how many cigarettes she smoked daily in the third trimester, and a recode designates whether she is a smoker.

Table A1: Year 2000 Standard Population for the United States (from the Compressed Mortality File description on age-adjustment).

Age	Number
All ages	274,633,642
Under 1 year	3,794,901
1-4 years	15,191,619
5-14 years	39,976,619
15-24 years	38,076,743
25-34 years	37,233,437
35-44 years	44,659,185
45-54 years	37,030,152
55-64 years	23,961,506
65-74 years	18,135,514
75-84 years	12,314,793
85 years and over	4,259,173

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Figure 1: Age-adjusted All Cause Mortality Rates per 100,000

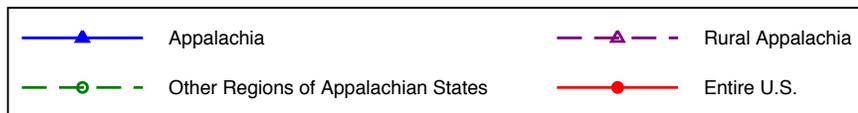
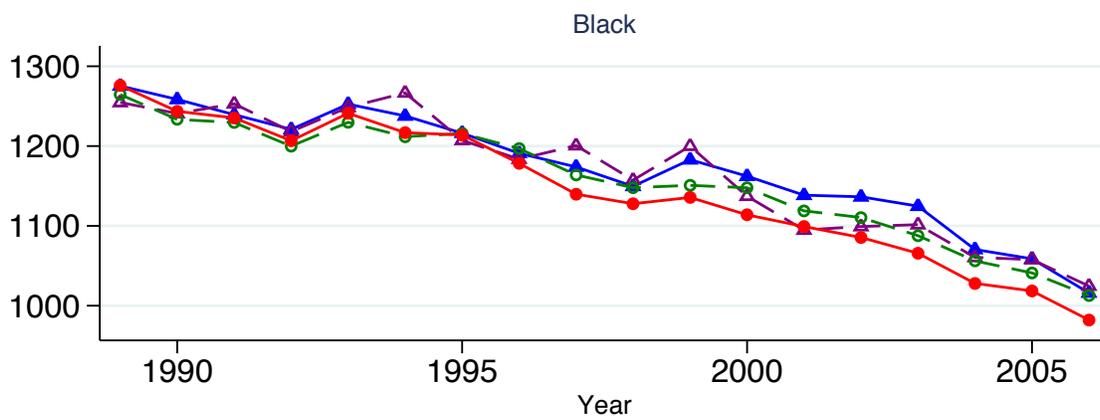
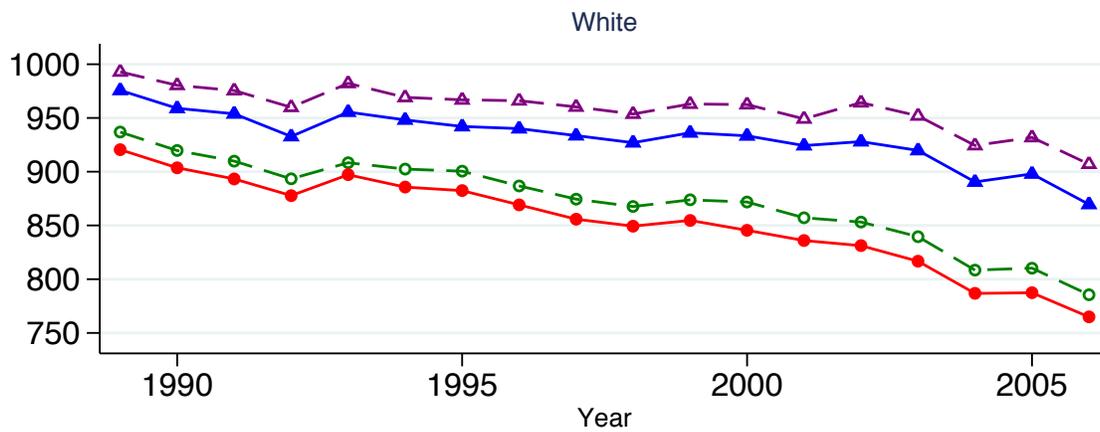
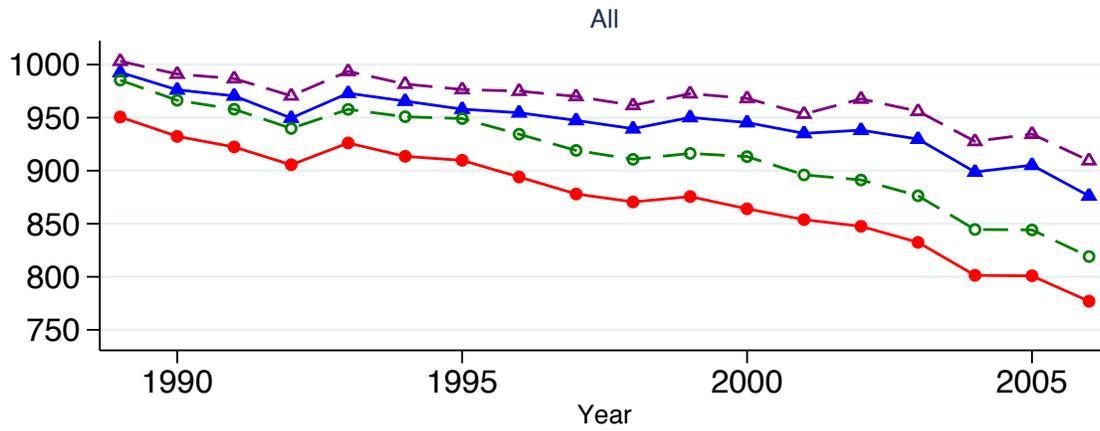


Figure 2: Infant Mortality per 100,000

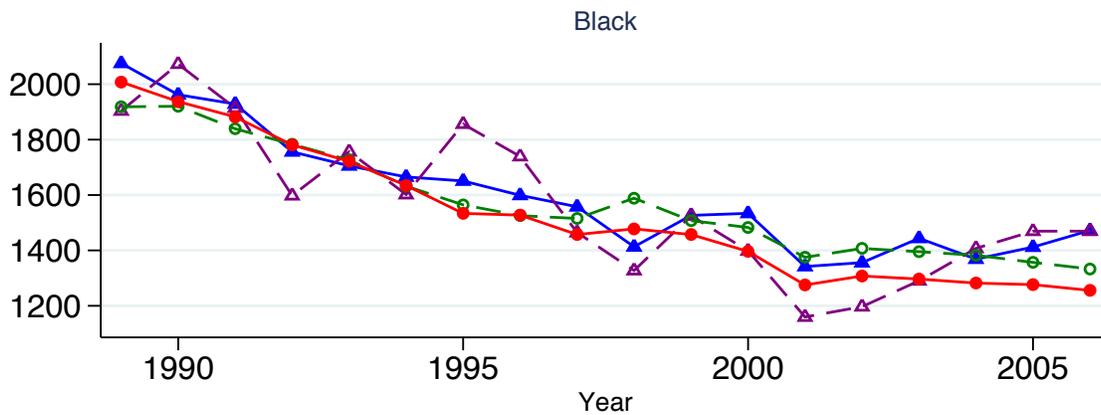
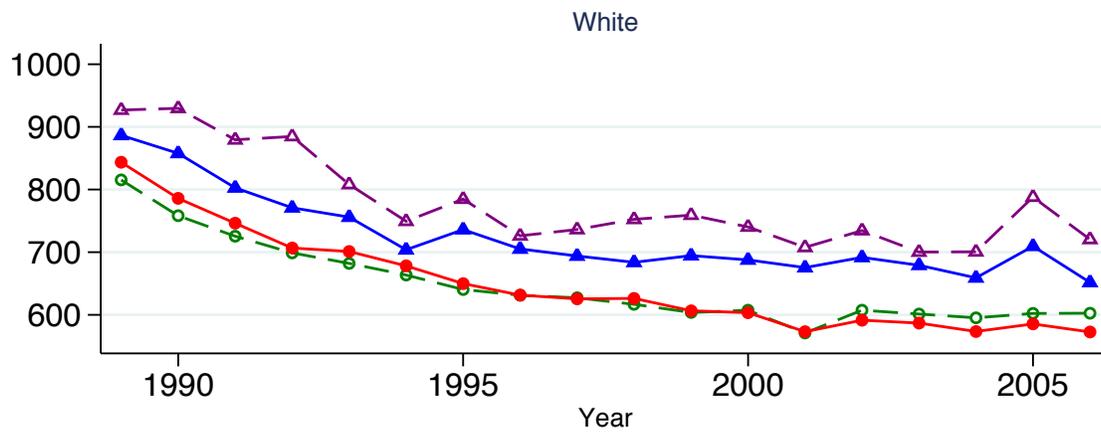
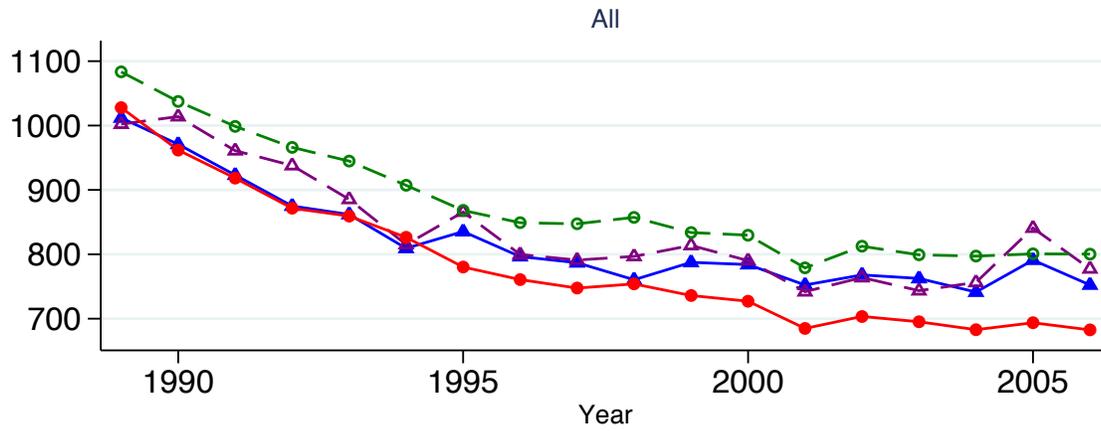


Figure 3: Mortality for Children 0 to 19 per 100,000

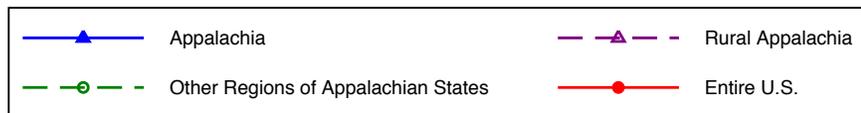
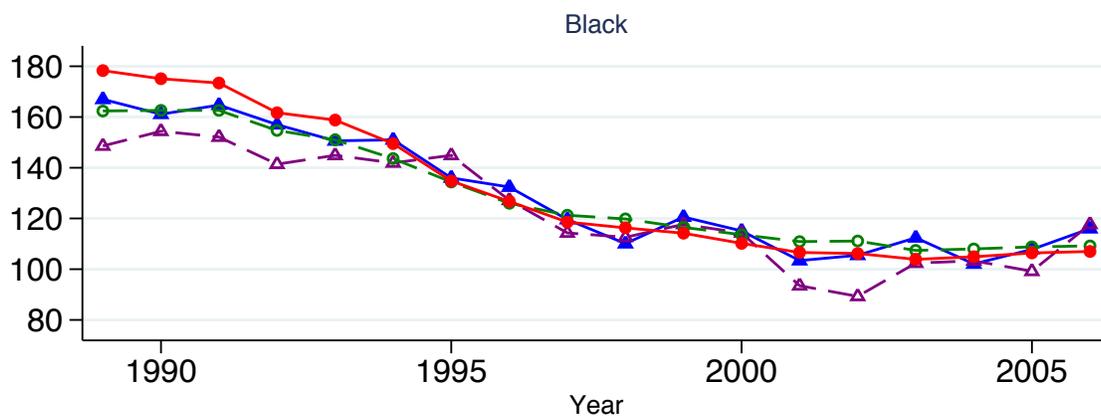
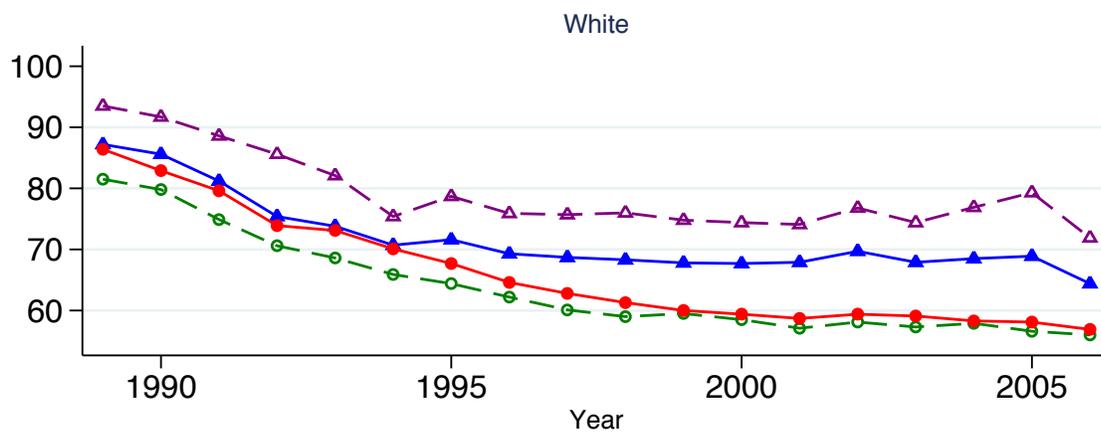
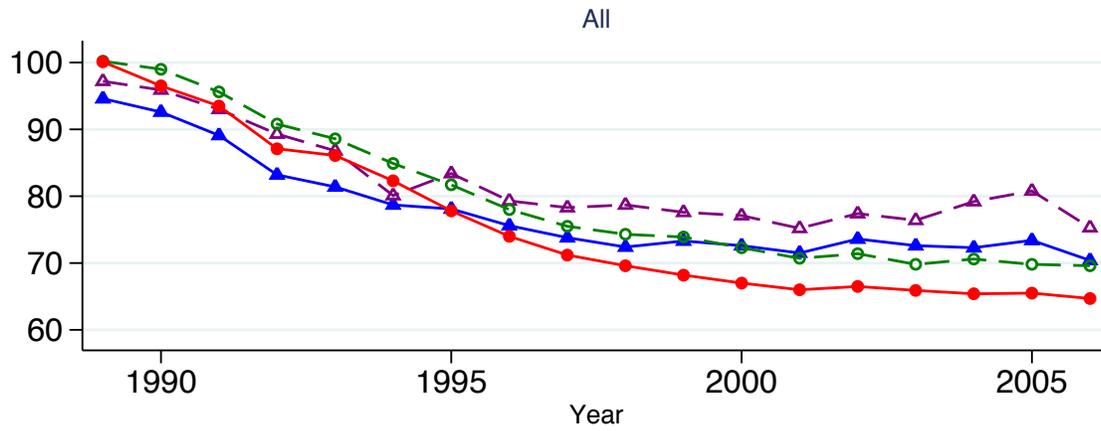


Figure 4: Low Birth Weight per 1,000 Live Births

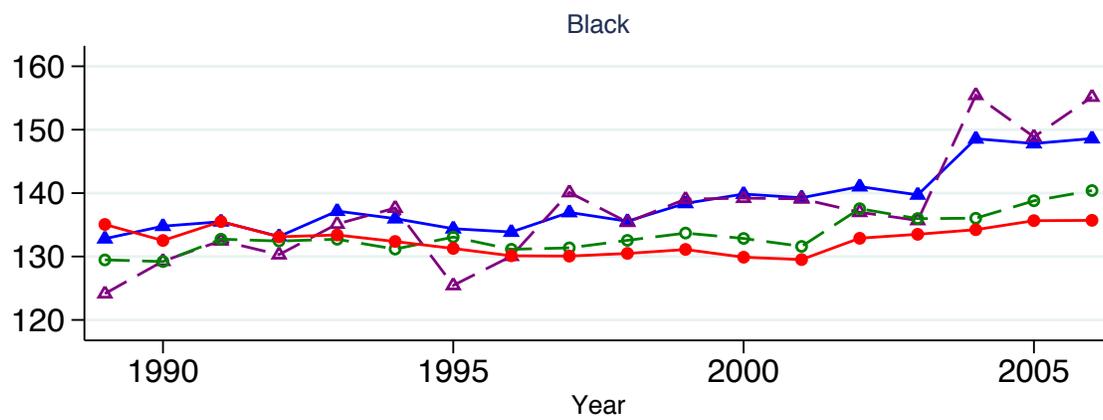
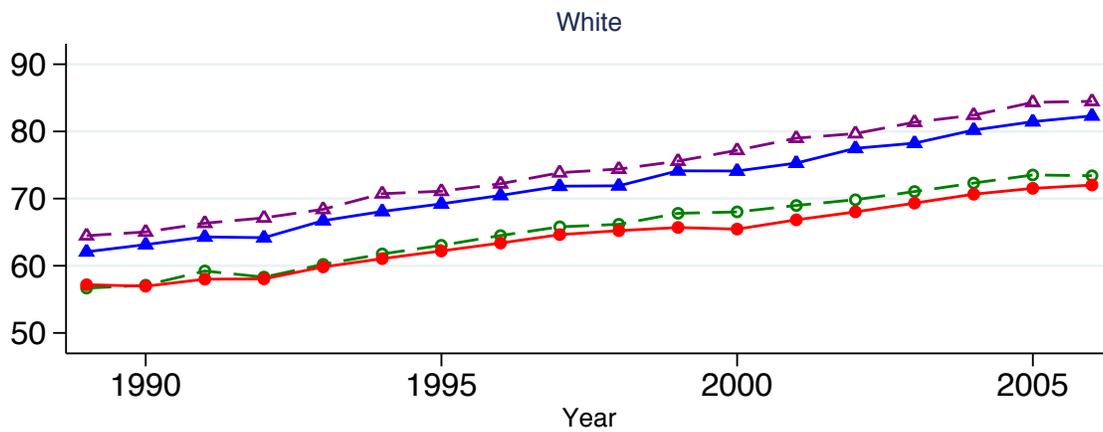
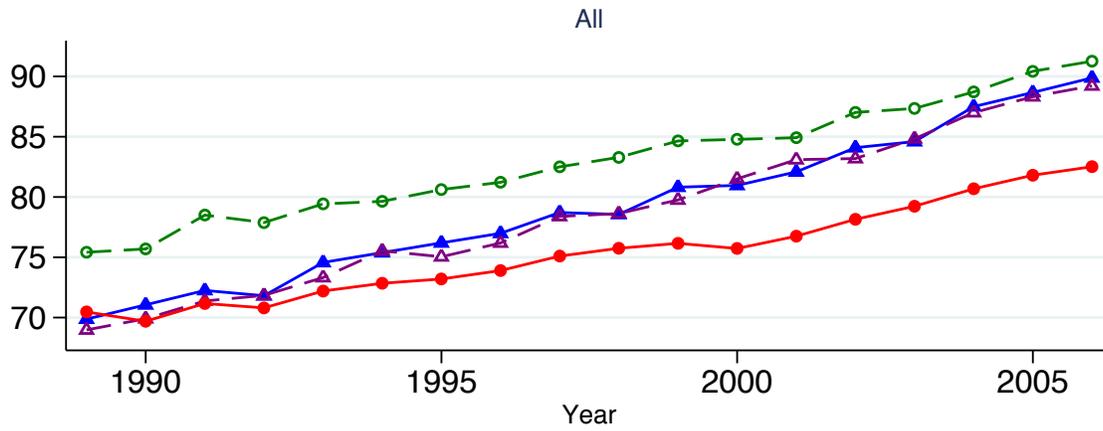


Figure 5: Percent of Infants Born Outside County of Residence

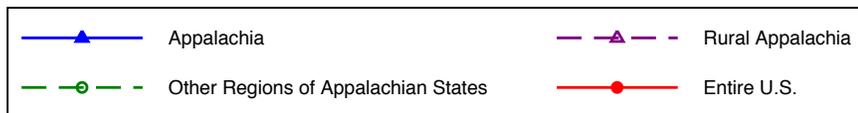
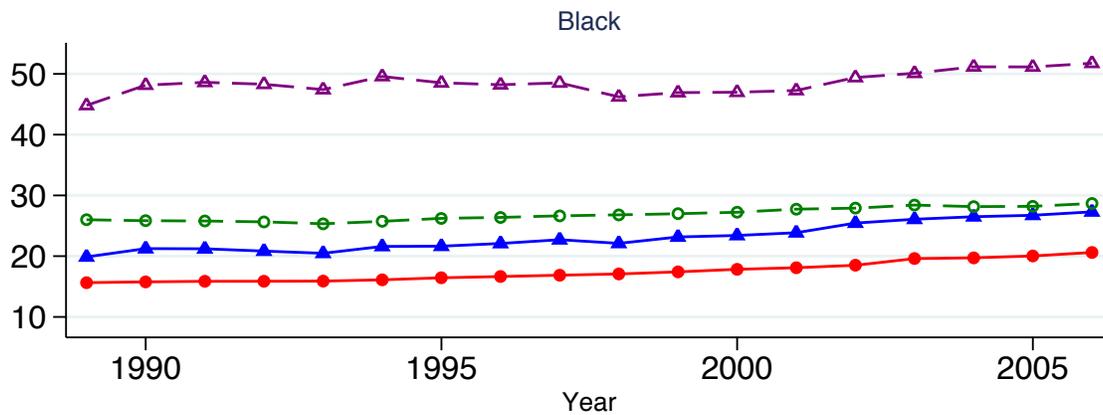
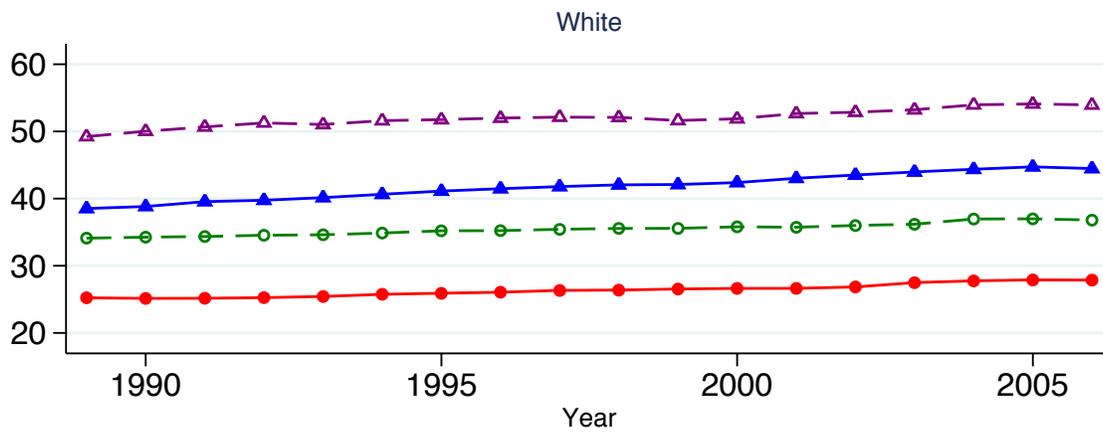
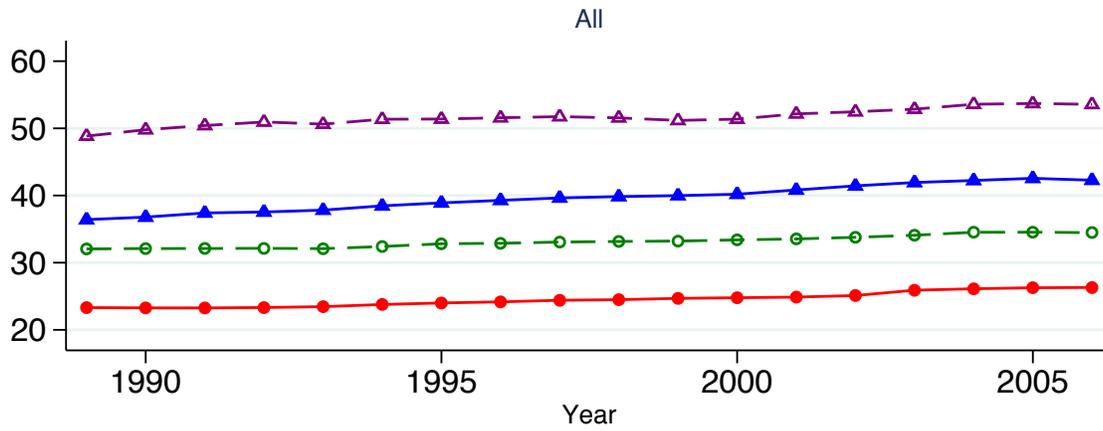


Figure 6: Percent of Infants Born in Hospitals

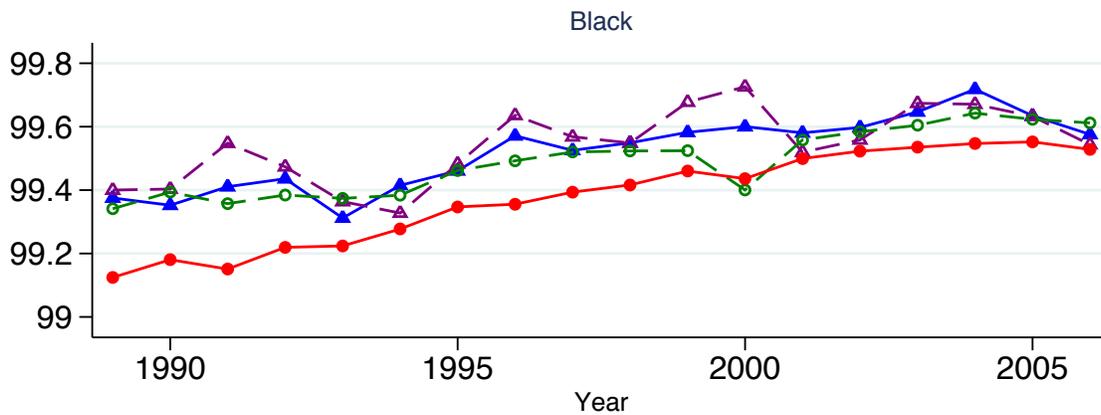
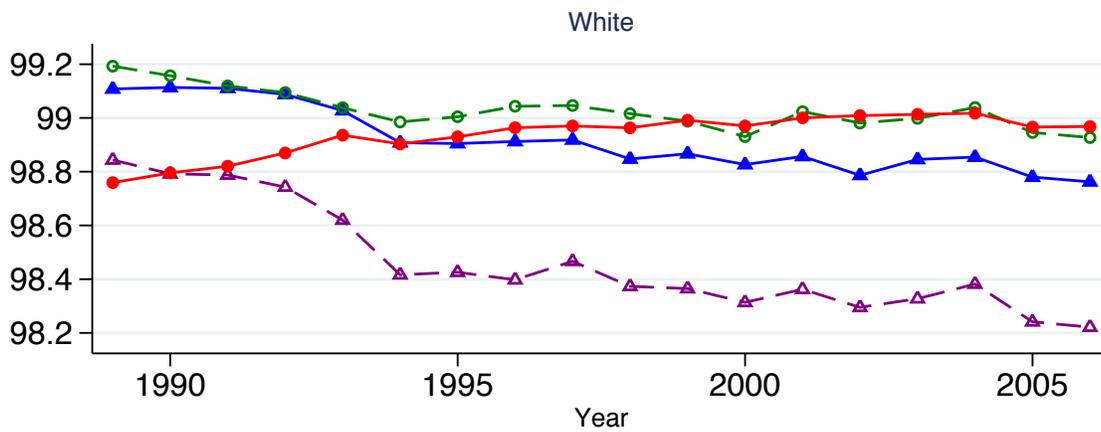
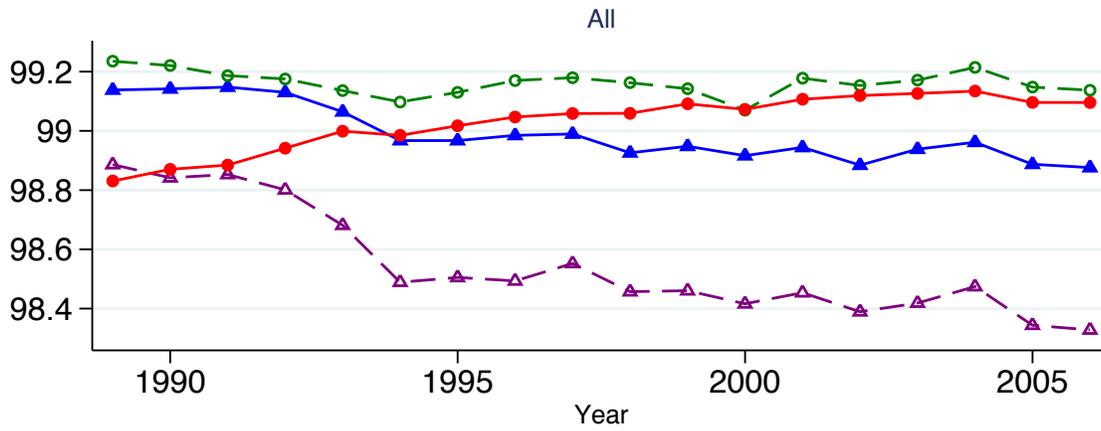


Figure 7: Percent of Infants Receiving Prenatal Care in First Trimester

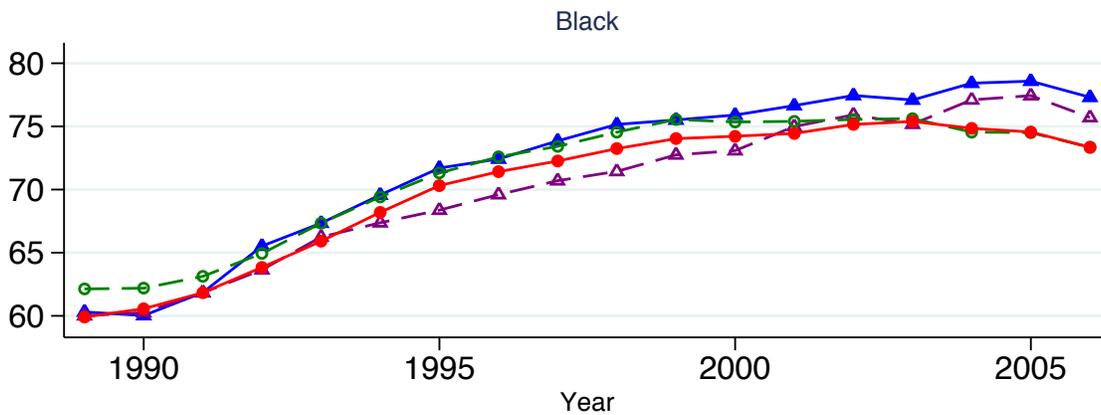
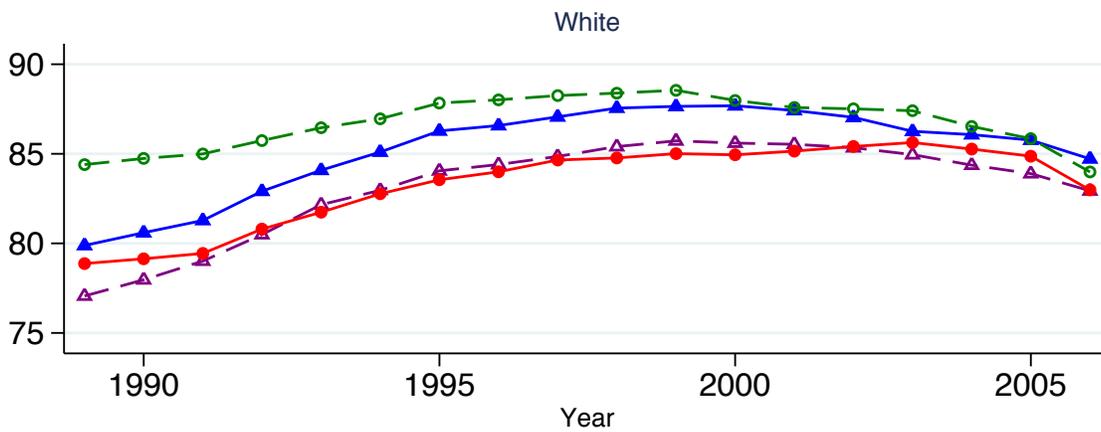
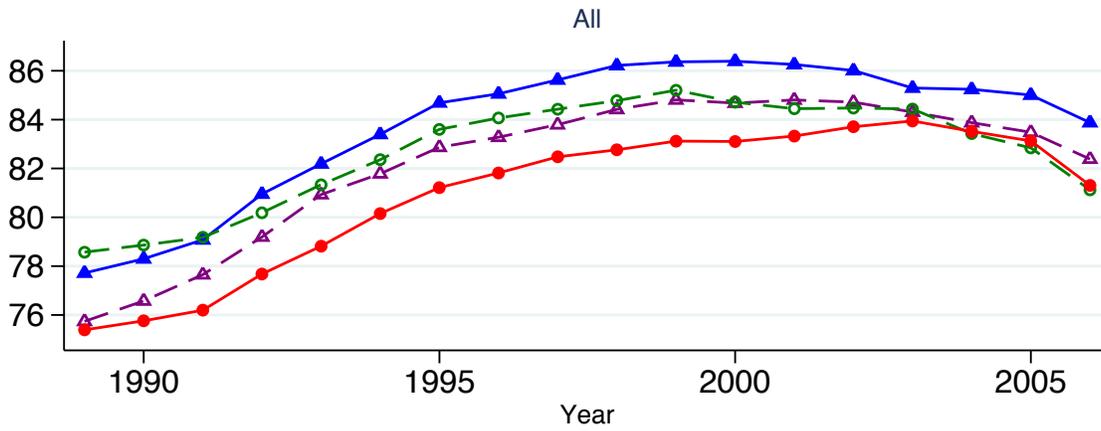


Figure 8: Percent of Infants Delivered by C-Section

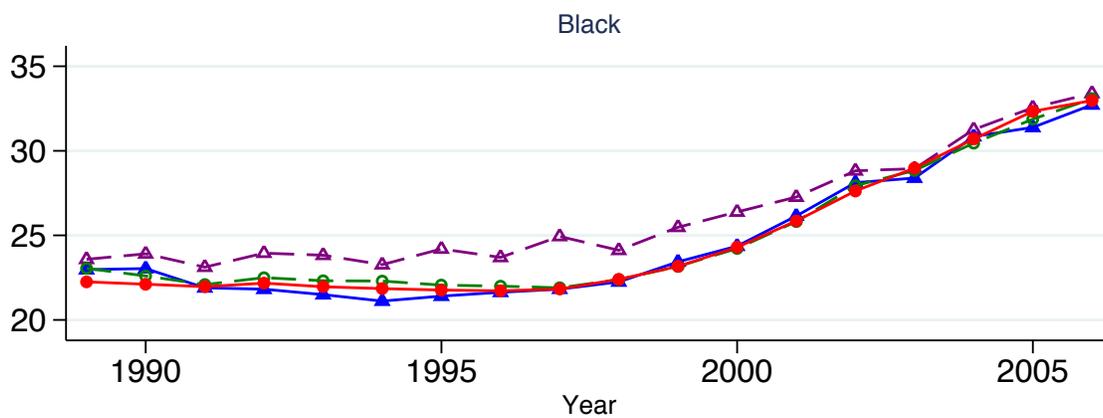
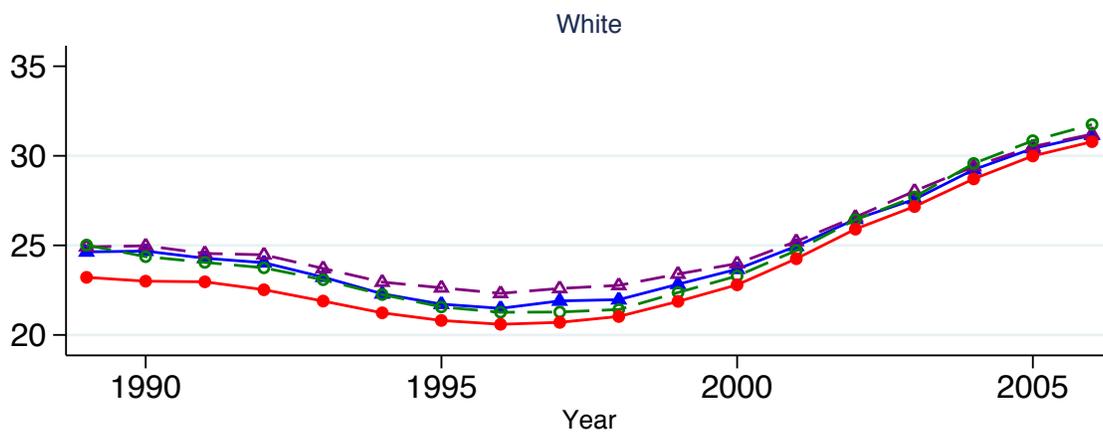
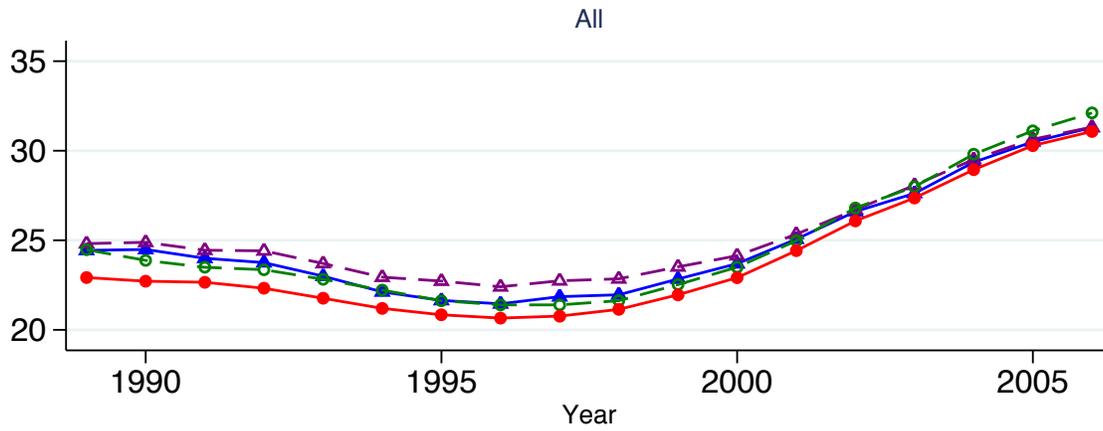


Figure 9: Percent of Mothers with Less than High School Education

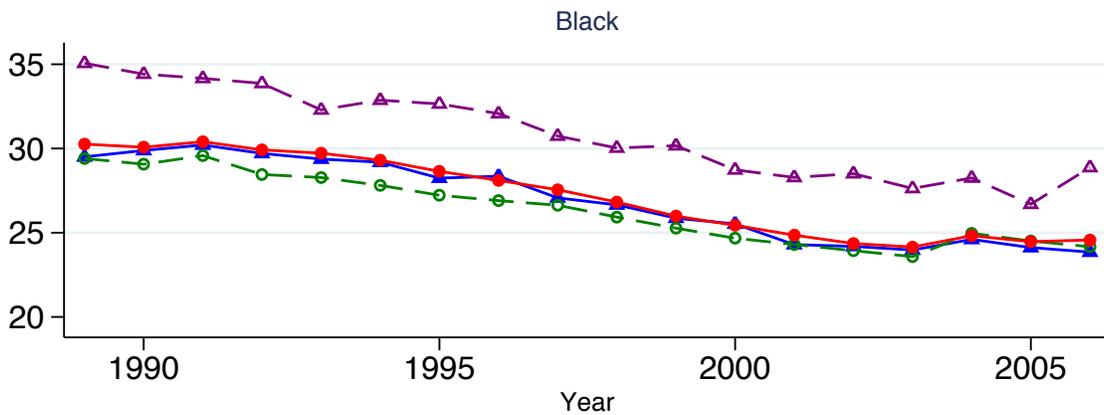
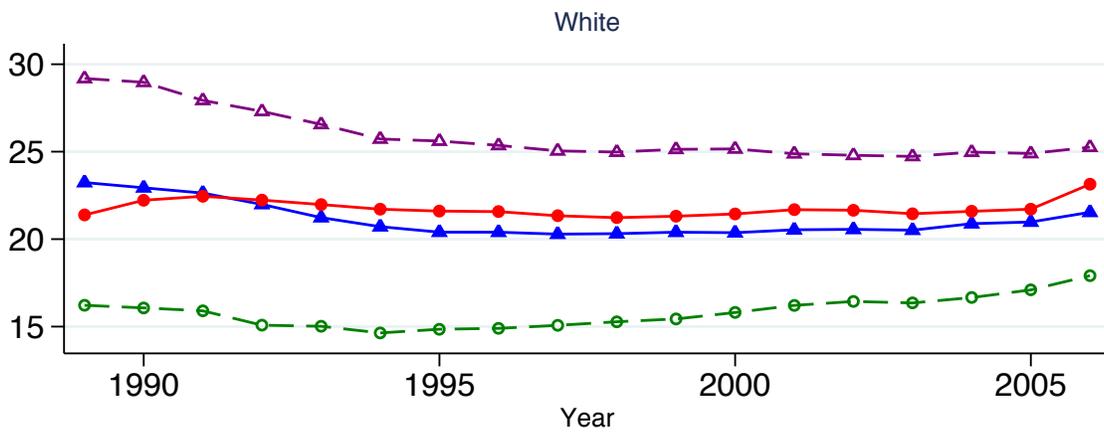
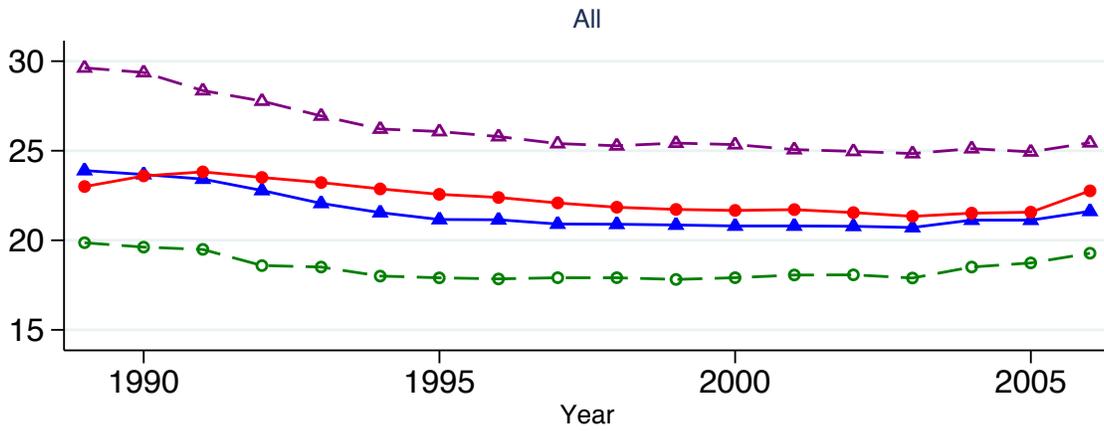


Figure 10: Percent of Mothers Less than 19 Years of Age

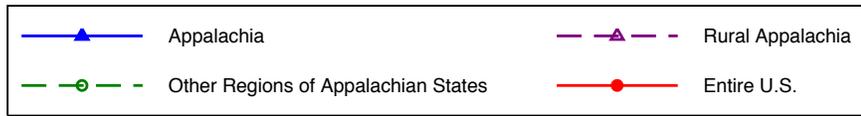
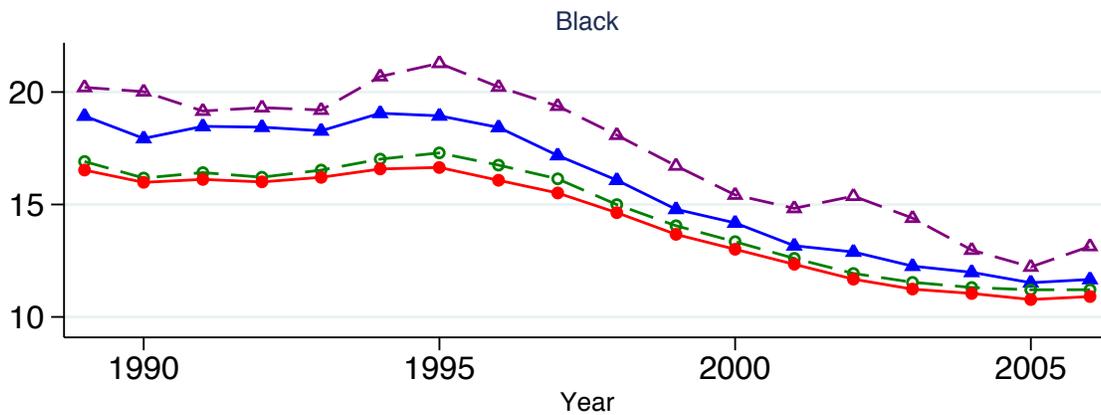
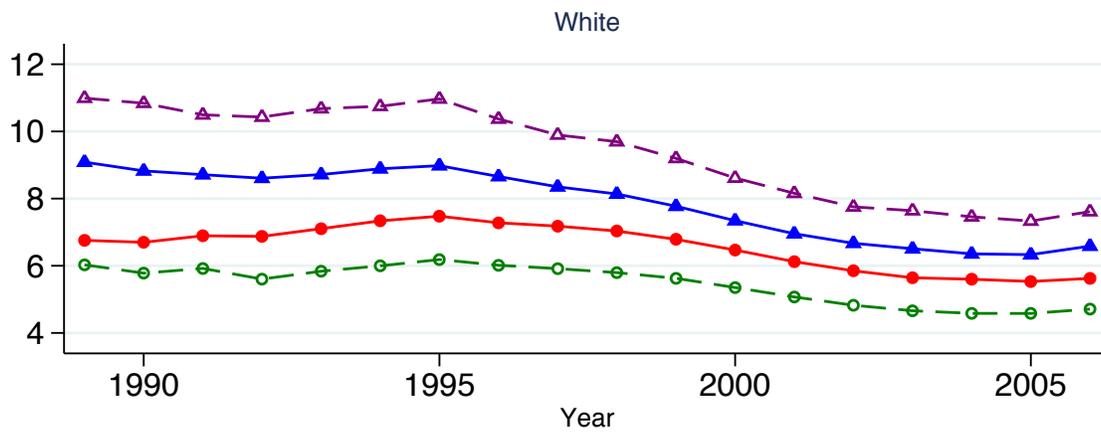
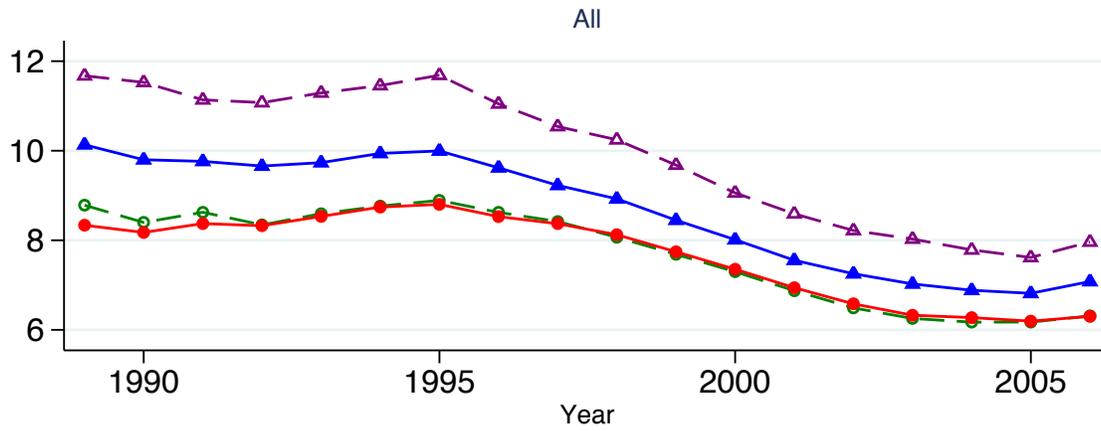


Figure 11: Percent of Mothers Smoking During Pregnancy

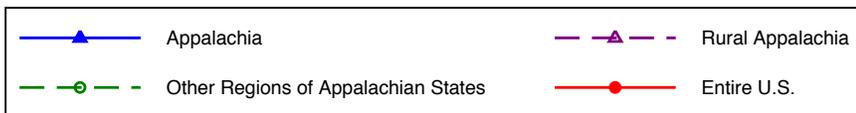
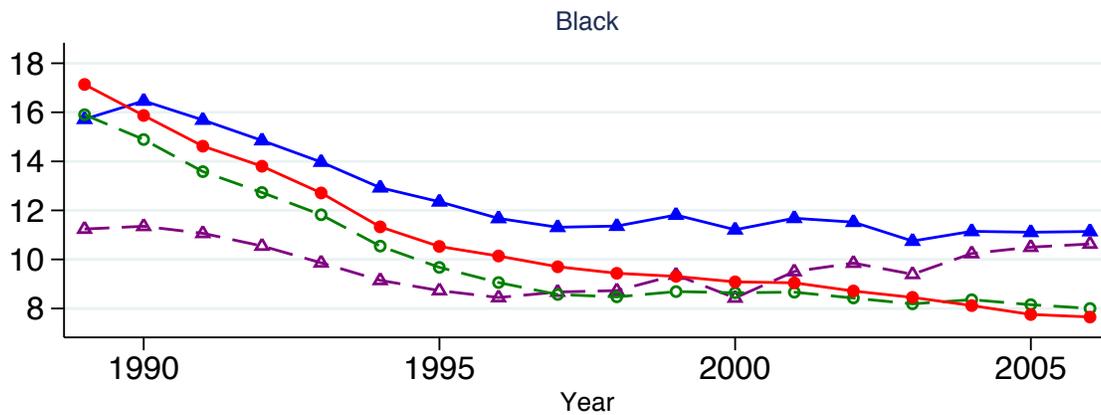
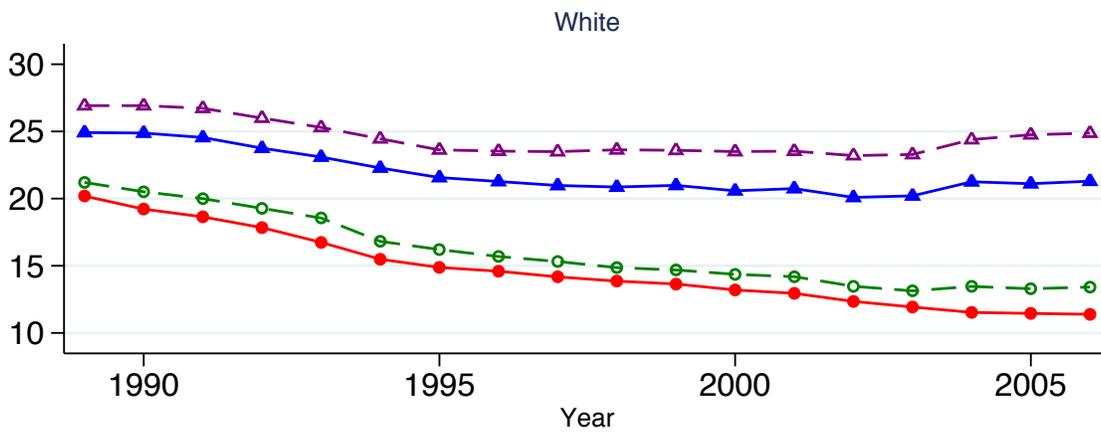
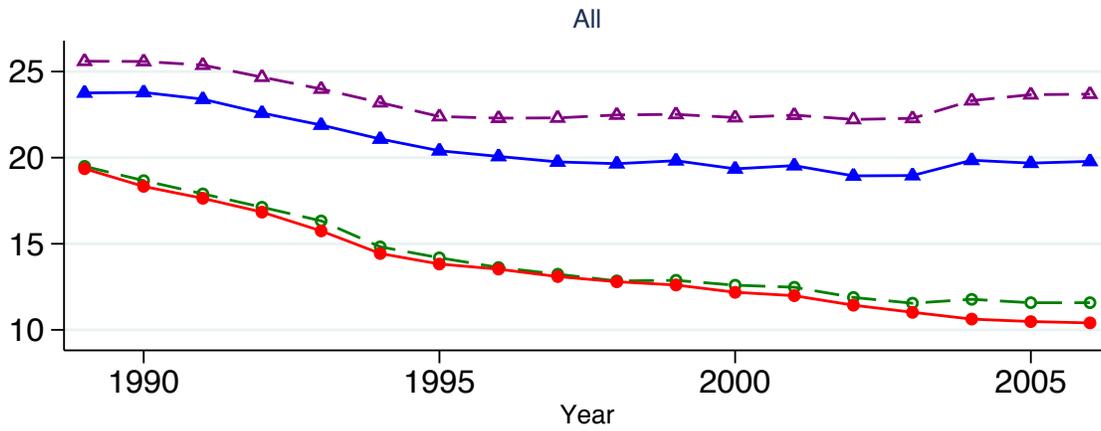


Figure 12: Percent of Mothers with Chronic Hypertension

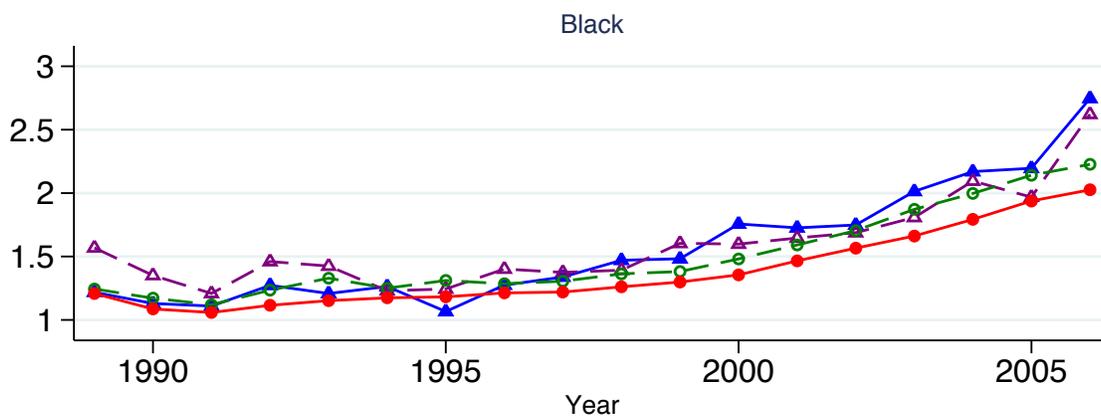
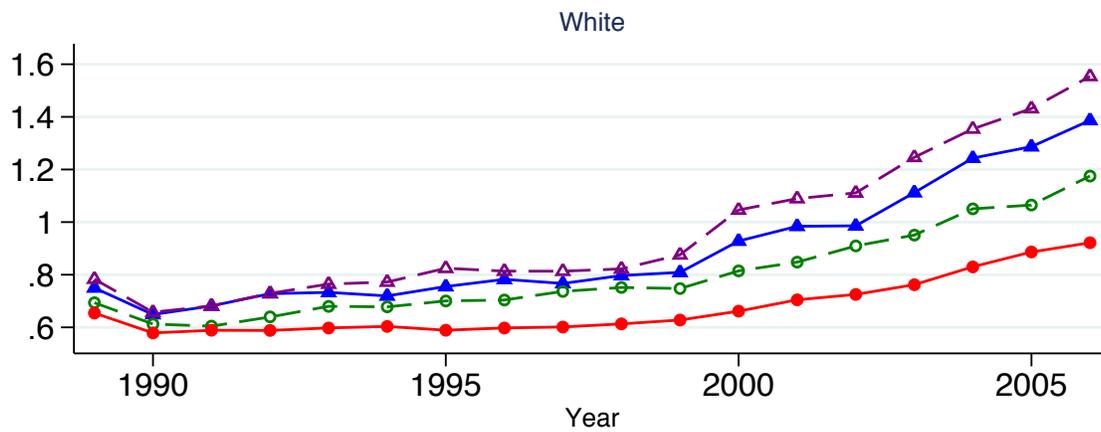
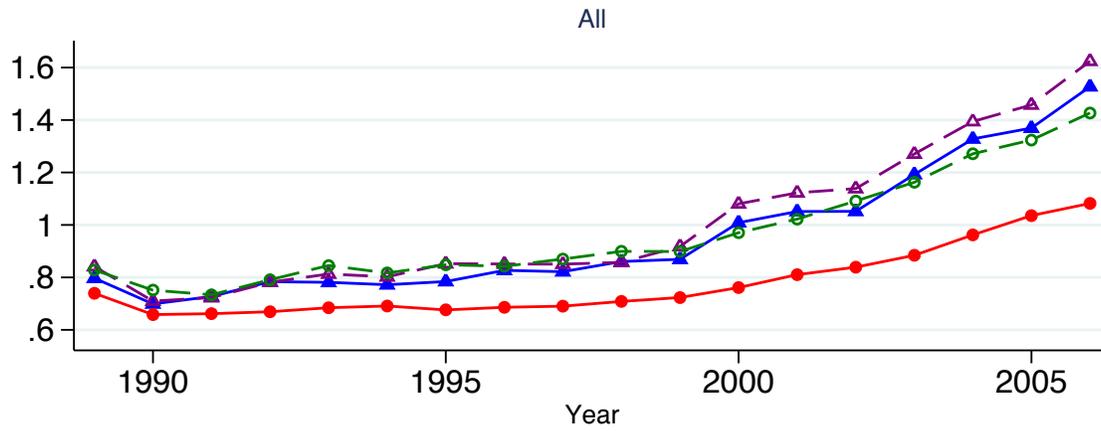


Figure 13: Age-adjusted Mortality from Cardiovascular Disease per 100,000

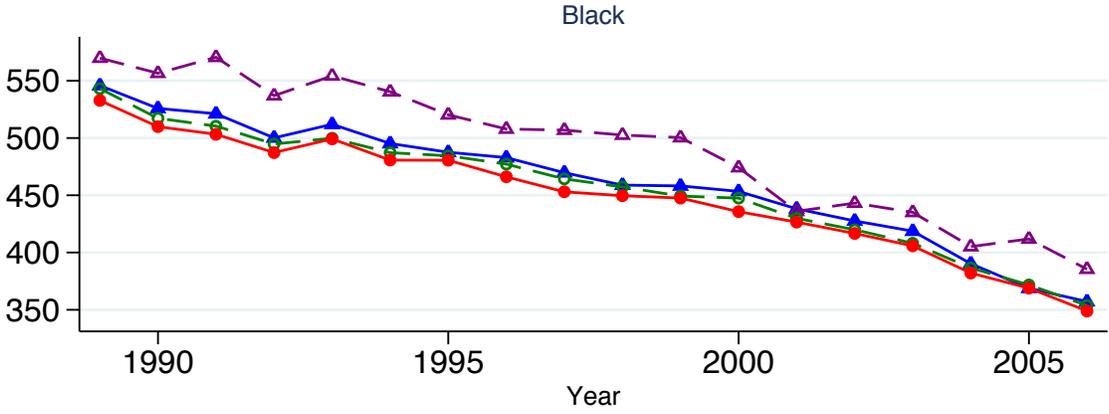
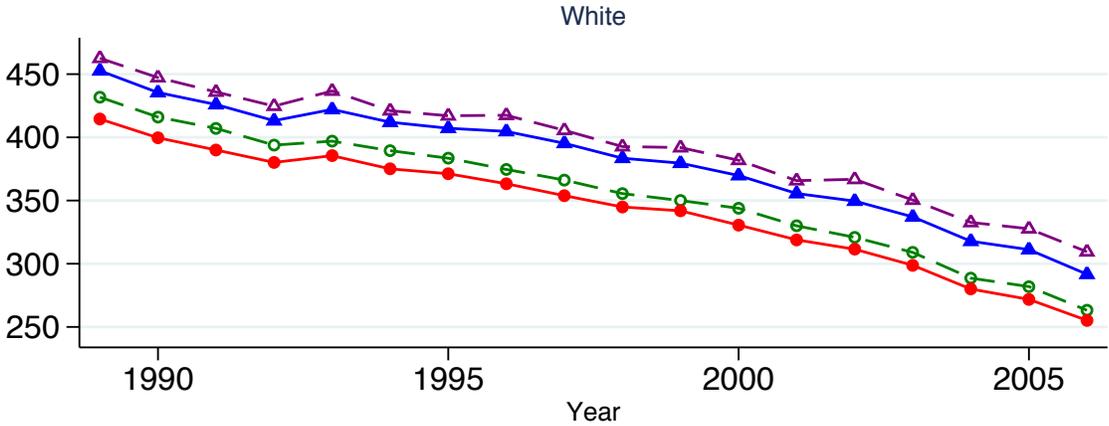
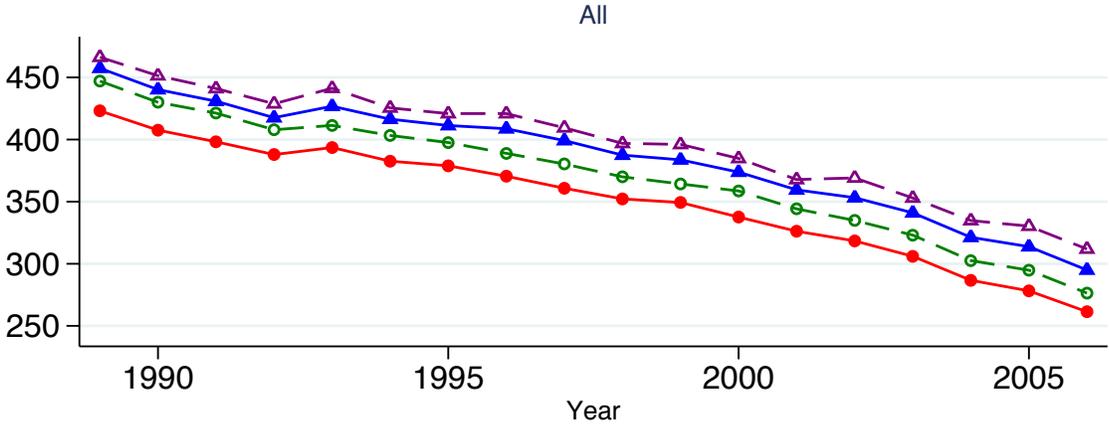


Figure 14: Age-adjusted Mortality from Cancer per 100,000

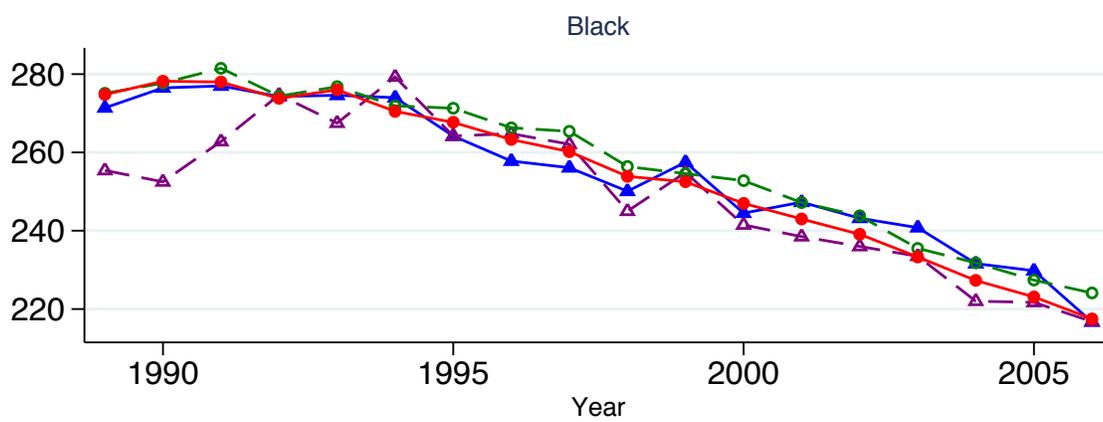
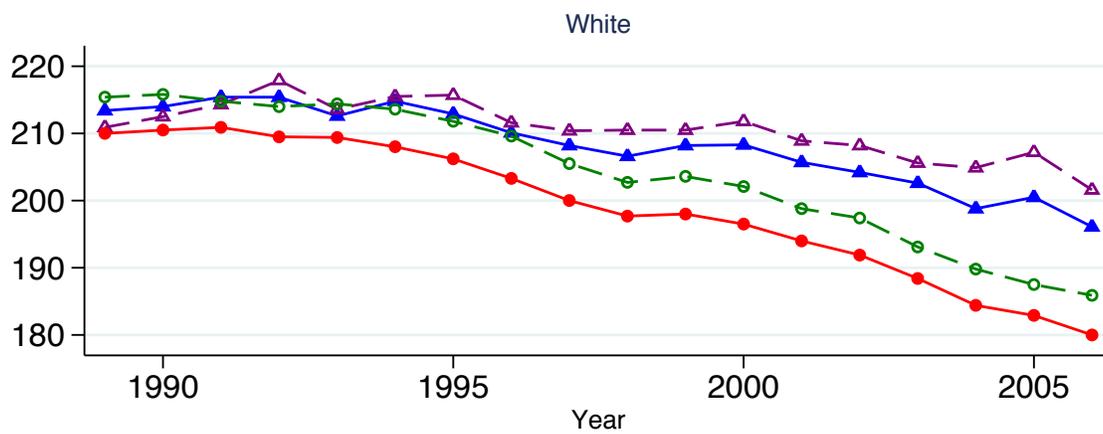
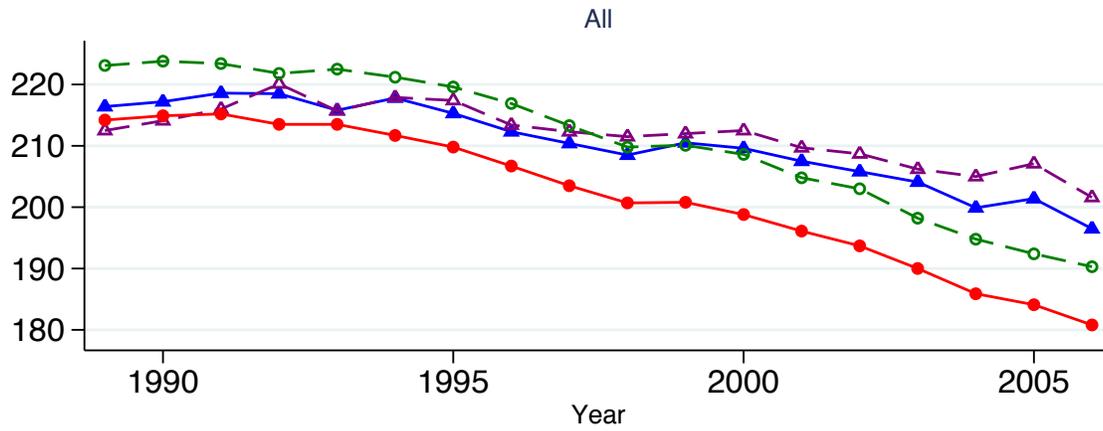


Figure 15: Age-adjusted Mortality from Diabetes per 100,000

